THE
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FEBRUARY 1957

THURSTON SCOTT WELTON, M.D., EDITOR

Symposium on the Esophagus and Its Diseases

Emil A. Naclerio, M.D., *Guest Editor*

SIXTY-SIXTH YEAR OF PUBLICATION

FEBRUARY 1957

THE American JOURNAL OF Surgery

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Contents • February 1957

VOLUME NINETY-THREE NUMBER TWO

SYMPOSIUM ON THE ESOPHAGUS AND ITS DISEASES

Foreword 153
EMIL A. NACLERIO

BASIC ASPECTS

Newer Concepts of the Physiology of Deglutition and Dysphagia 154
NICHOLAS C. HIGHTOWER, JR.

Roentgen Differential Diagnosis of Lesions Affecting the Esophagus. 163
R. VINCENT GRIECO AND NOEL F. BARTONE

Esophagoscopy in the Diagnosis and Treatment of Esophageal Diseases . . . 183
MAX L. SOM AND LEON M. ARNOLD

CONGENITAL DISEASES

Congenital Anomalies of the Esophagus 196
ROBERT R. SHAW AND DONALD L. PAULSON

DIVERTICULA

Pharyngoesophageal Diverticulum 205
KENNETH W. WARREN

Contents Continued on Page 5

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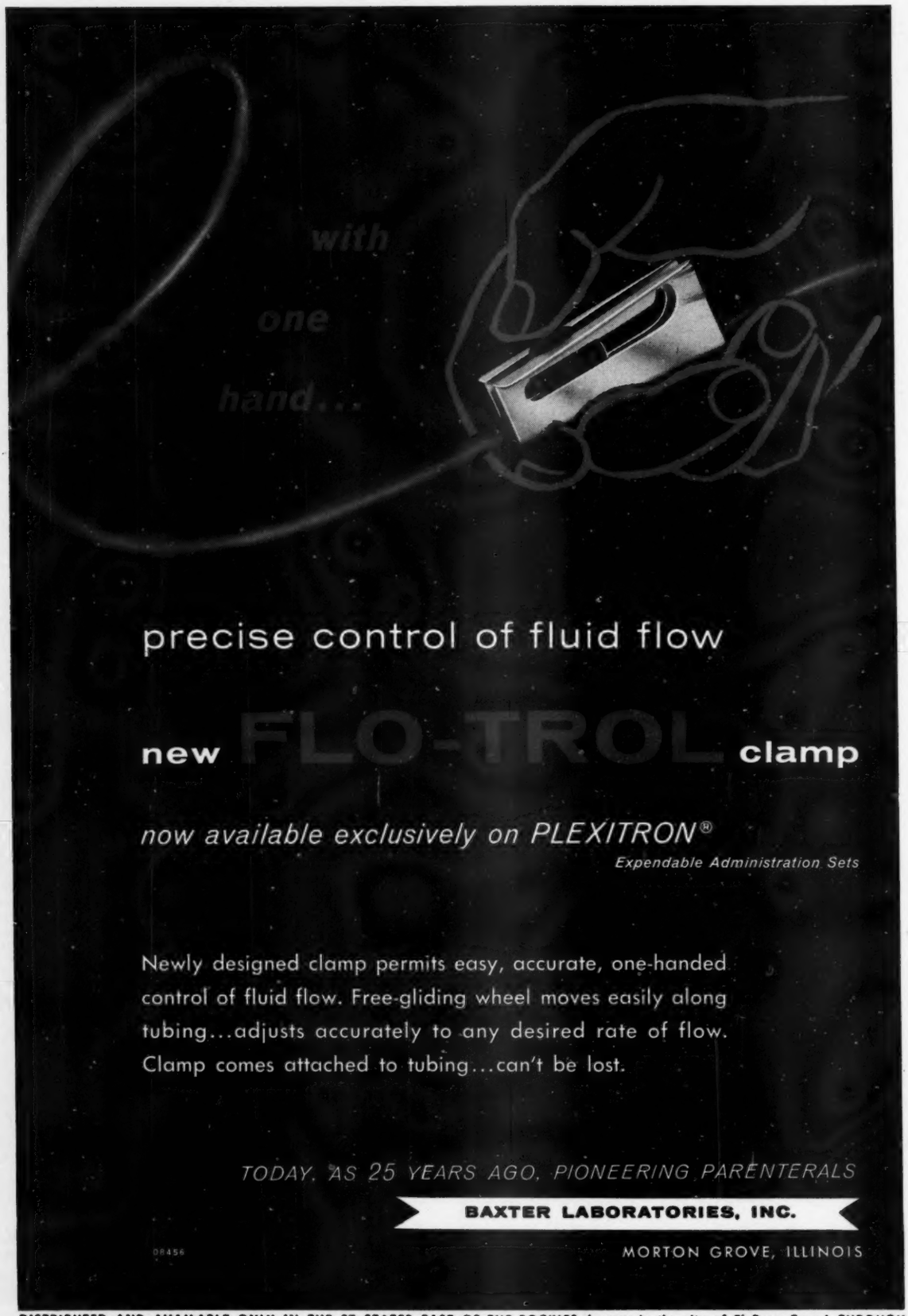

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Contents • February 1957

VOLUME NINETY-THREE NUMBER TWO

Diverticula of the Thoracic Esophagus 218
EMIL A. NACLERIO

ESOPHAGITIDES, STRICTURES AND WEBS

Reflux Esophagitis 228
HARRY W. HALE, JR. AND THEODORE DRAPANAS

Peptic Esophagitis with Duodenal Ulcer 234
ASHER WINKELSTEIN

Peptic Stenosis of Esophagus 238
EDWARD B. BENEDICT AND GEORGE L. NARDI

Corrosive Esophagitis 242
JOHN F. DALY AND JOHN C. CARDONA

Lower Esophageal Web. 248
WALTER F. BUGDEN

TUMORS

Benign Tumors and Cysts of the Esophagus. 252
DAVID P. BOYD AND LUCIUS D. HILL, III

Carcinoma of the Esophagus 259
WILLIAM L. WATSON AND JOHN T. GOODNER

Endoscopic Intubation and Dilatation in the Management of Esophageal Carcinoma 266
MAX G. CARTER

RUPTURES AND PERFORATIONS

Rupture of the Esophagus 271
J. MAXWELL CHAMBERLAIN AND W. GRIMES BYERLY

Spontaneous Rupture of the Esophagus 282
ROBERT L. ANDERSON

Contents Continued on Page 7

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Contents • February 1957

VOLUME NINETY-THREE NUMBER TWO

The "V Sign" in the Diagnosis of Spontaneous Rupture of the Esophagus (An Early Roentgen Clue) 291
EMIL A. NACLERIO

ACHALASIA (CARDIOSPASM)

Cardiospasm (Achalasia of the Cardia) 299
ARTHUR M. OLSEN, F. HENRY ELLIS, JR. AND BRIAN CREAMER

FOREIGN BODIES

Foreign Bodies in the Esophagus 308
CHEVALIER L. JACKSON

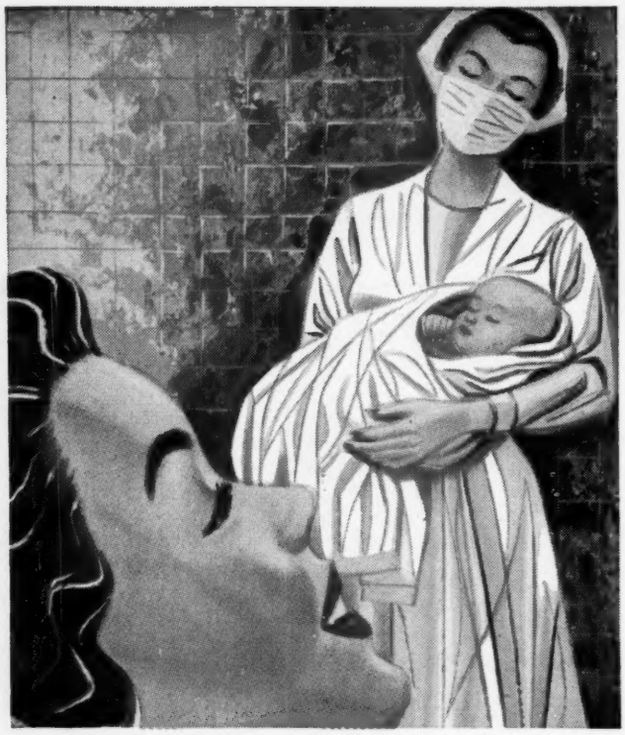
VARICES AND FISTULAS

Bleeding Esophageal Varices 313
CORNELIUS E. SEDGWICK

Acquired Non-malignant Esophagorespiratory Fistula 321
FRANK PHILIP COLEMAN

ESOPHAGEAL INTUBATION

Intubation of the Esophagus. Its Role in Preventing Aspiration Pneumonia and Asphyxial Death 329
JOSEPH G. GIUFFRIDA AND DANTE BIZZARRI



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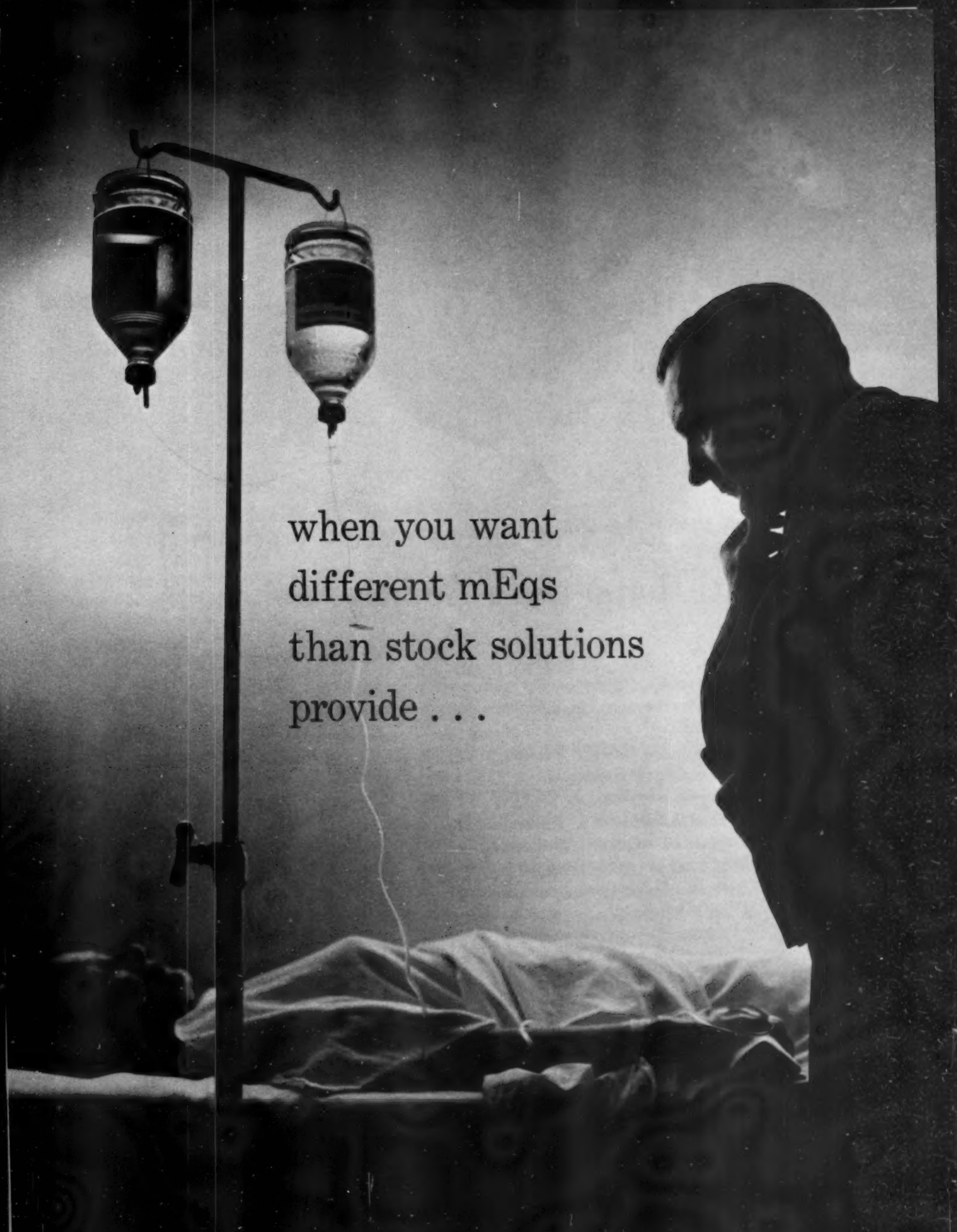
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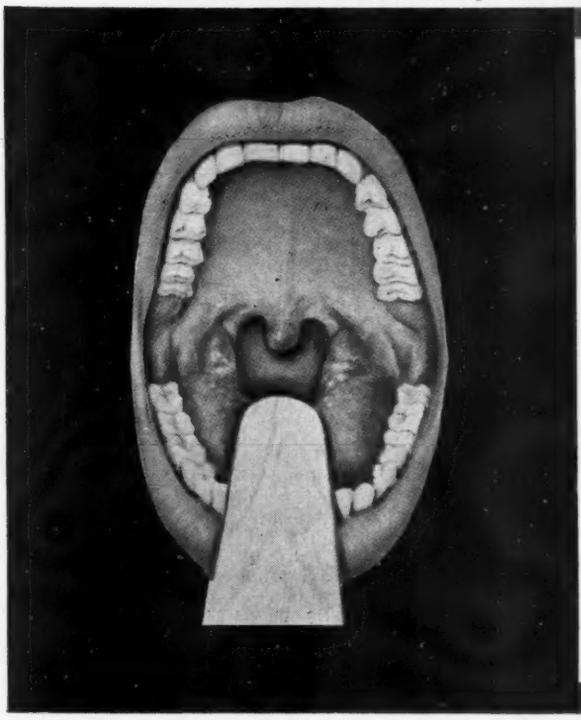

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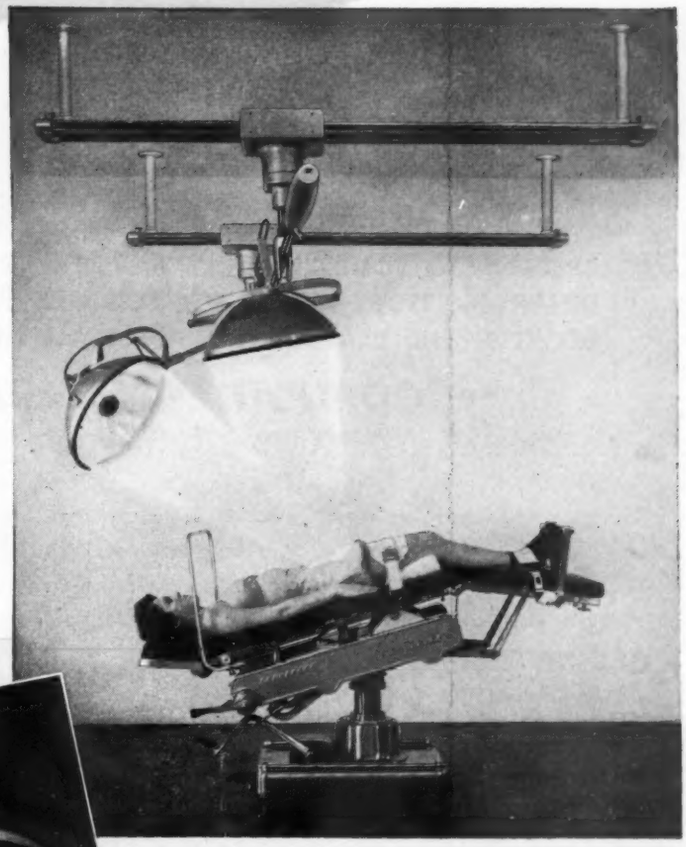

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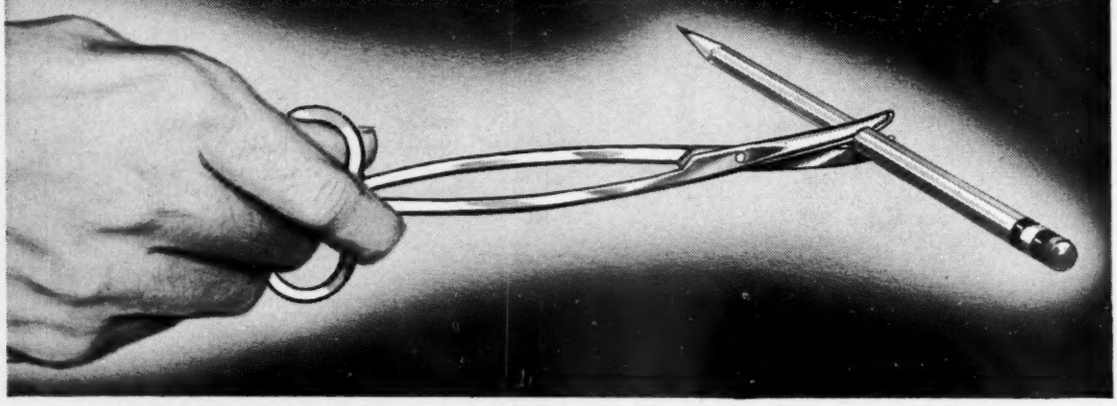

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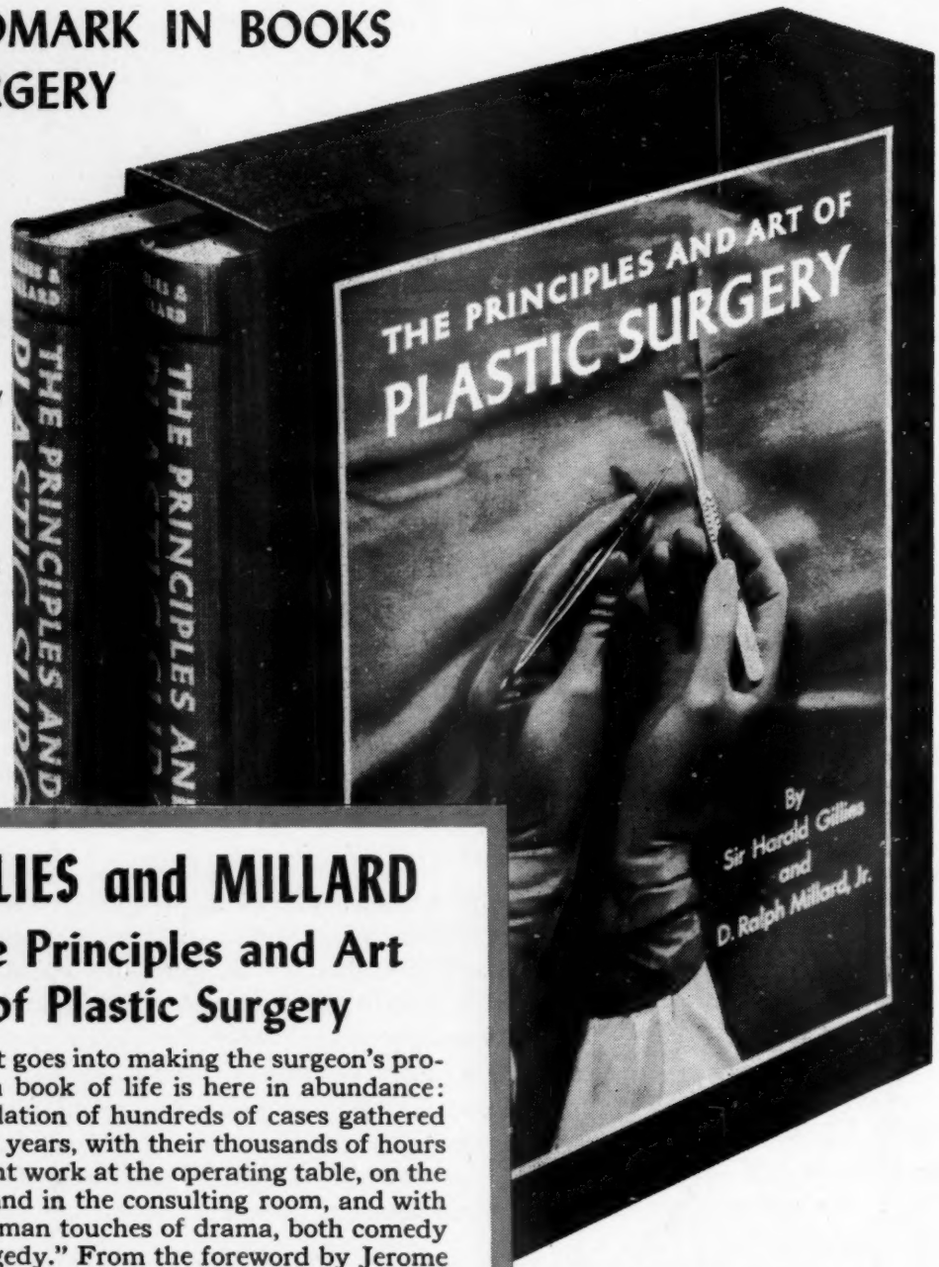

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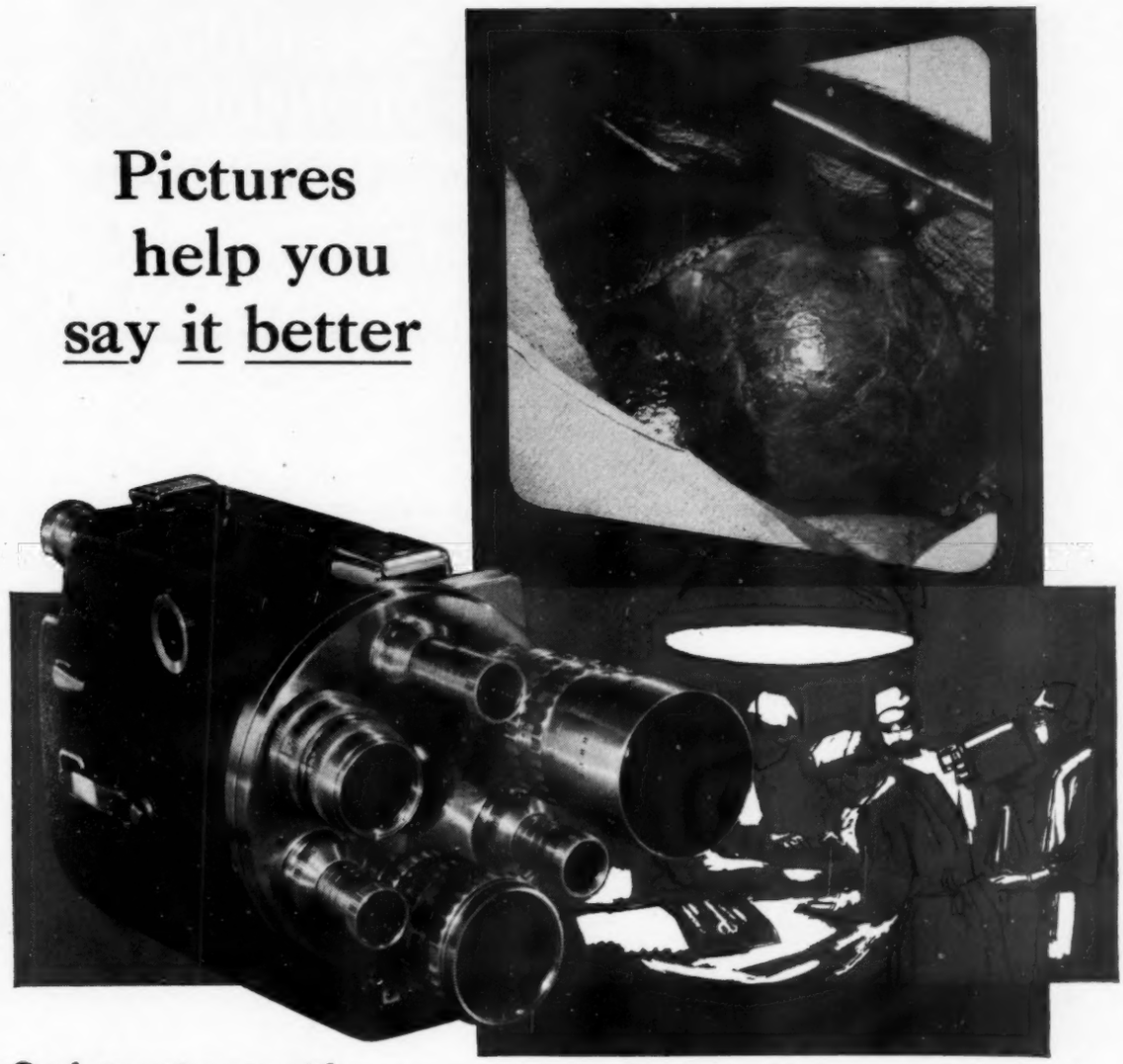

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[1] Miller, J. M.; Surmonte, J. A.; Ginsberg, M., and Ablondi, F. B.: Streptokinase Intramuscularly in the Treatment of Infection and Edema. (Scientific Exhibit) *Postgraduate Medicine* Vol. 20, No. 3: 260-267 (Sept.) 1956.

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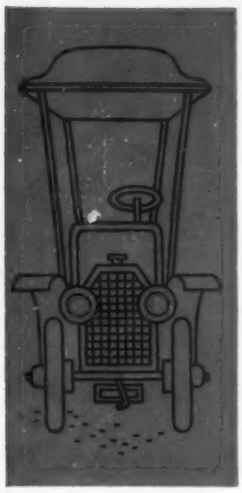

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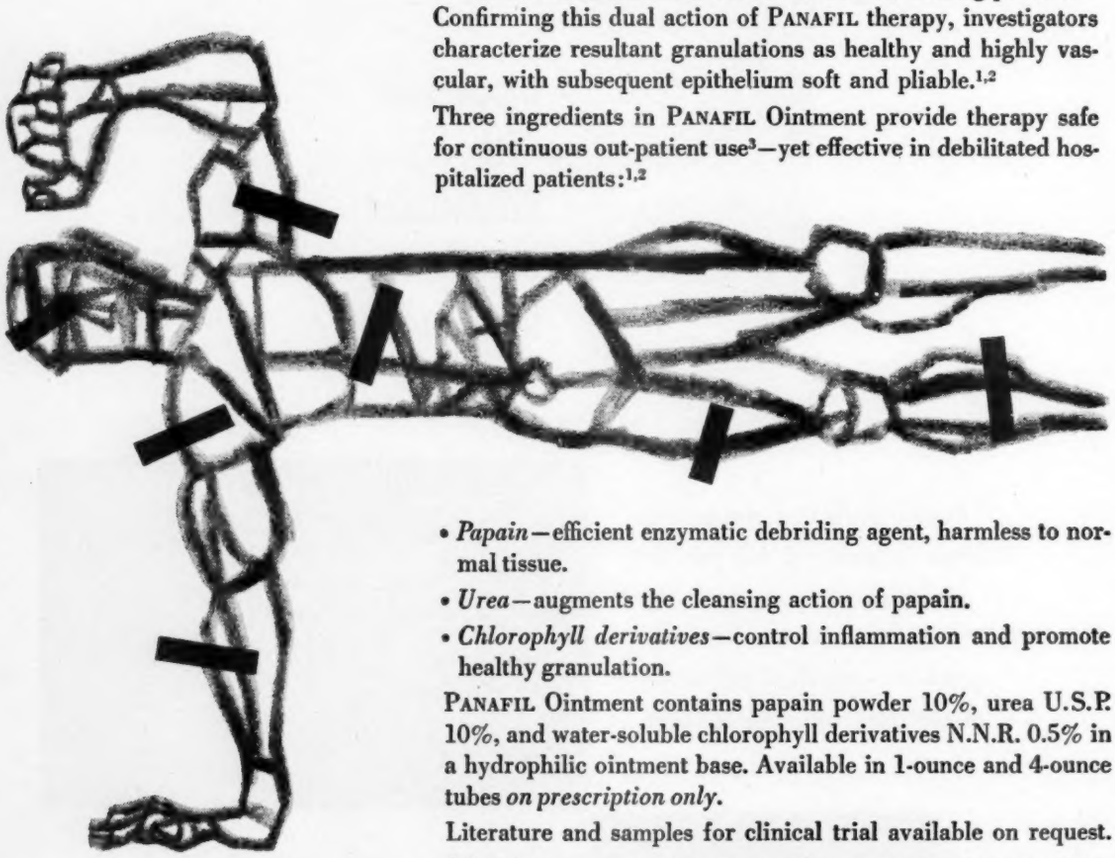

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(1) Miller, E. W.: New York State J. Med. *56*:1446, 1956. (2) Morrison, J. E., and Casali, J. L.: Am. J. Surg., to be published. (3) Garnes, A. L., and Barnard, R. D.: Angiology, in press.

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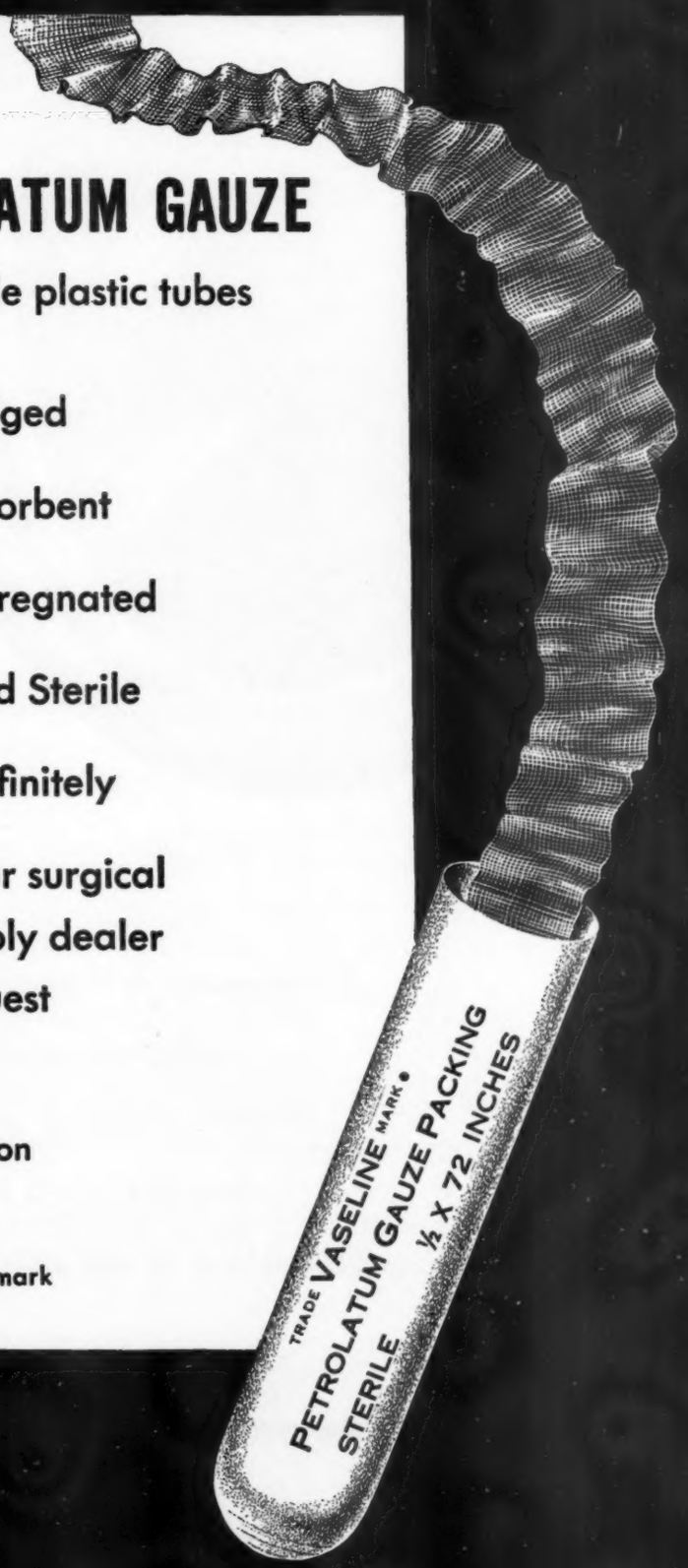

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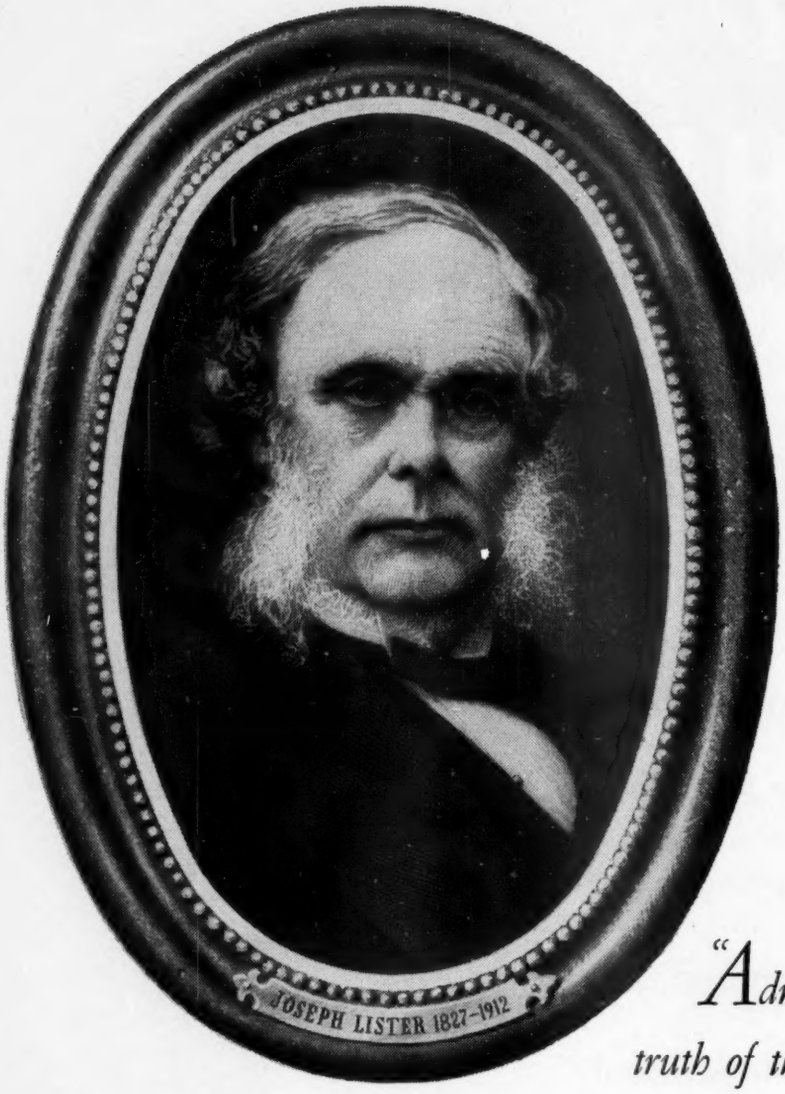

"Admitting then, the truth of the germ theory, and proceeding in accordance with it, we must, when dealing with any case, destroy in the first instance once for all any septic organisms which may exist within the part concerned; and after this has been done, our efforts must be directed to the ***prevention of the entrance of others into it."***

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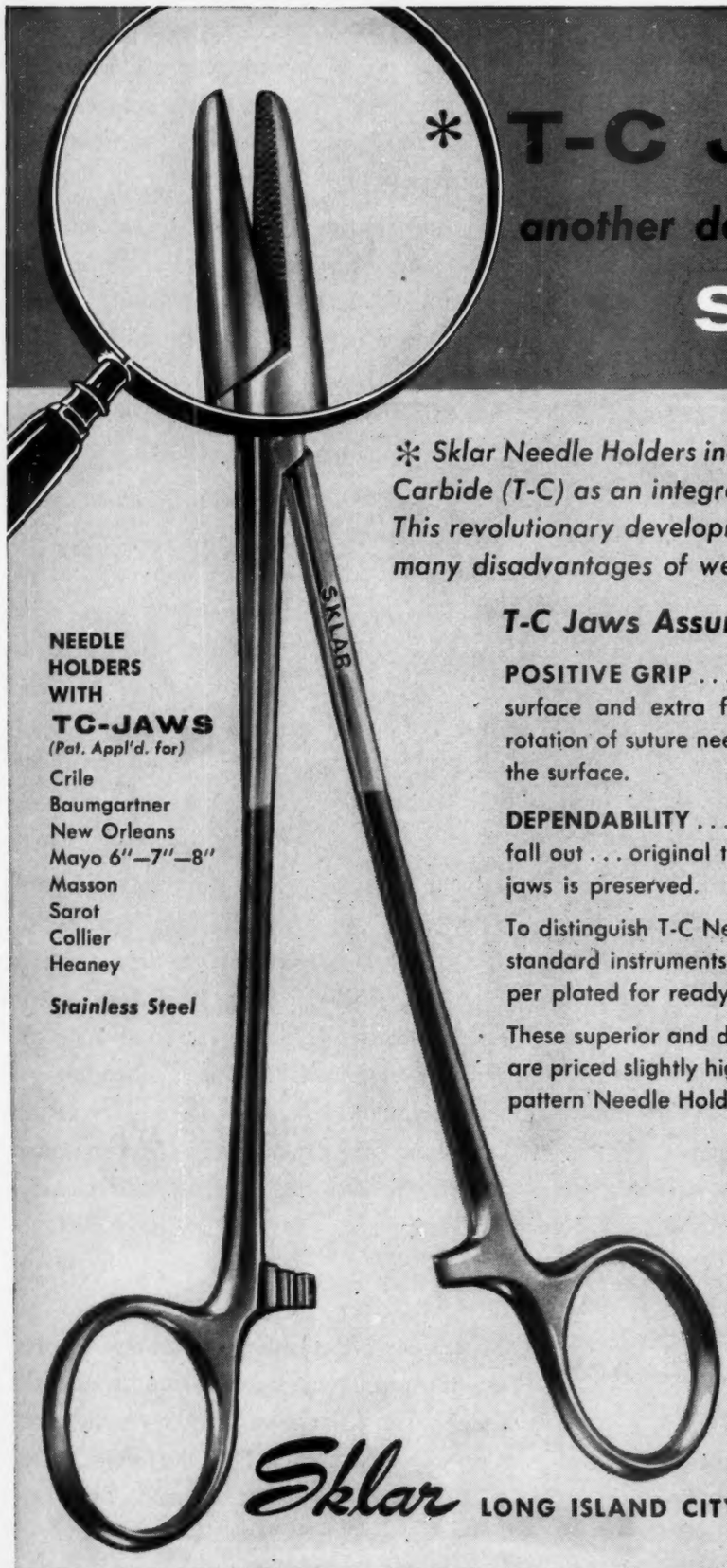

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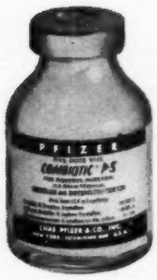

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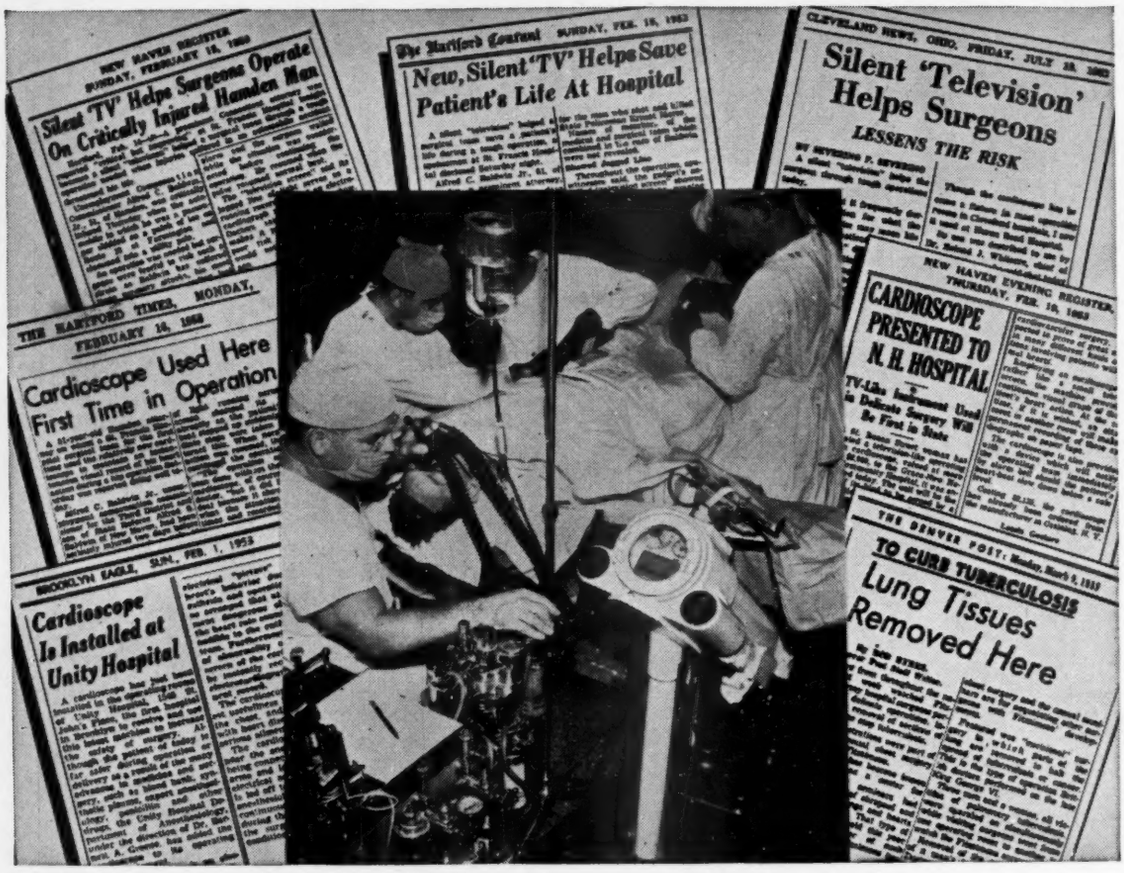
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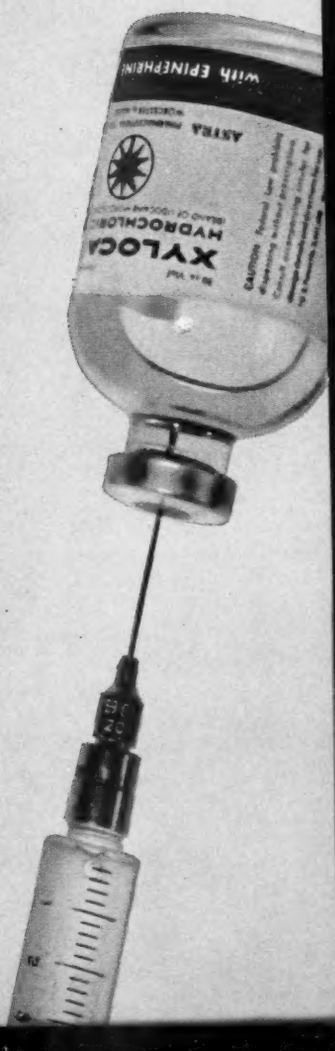

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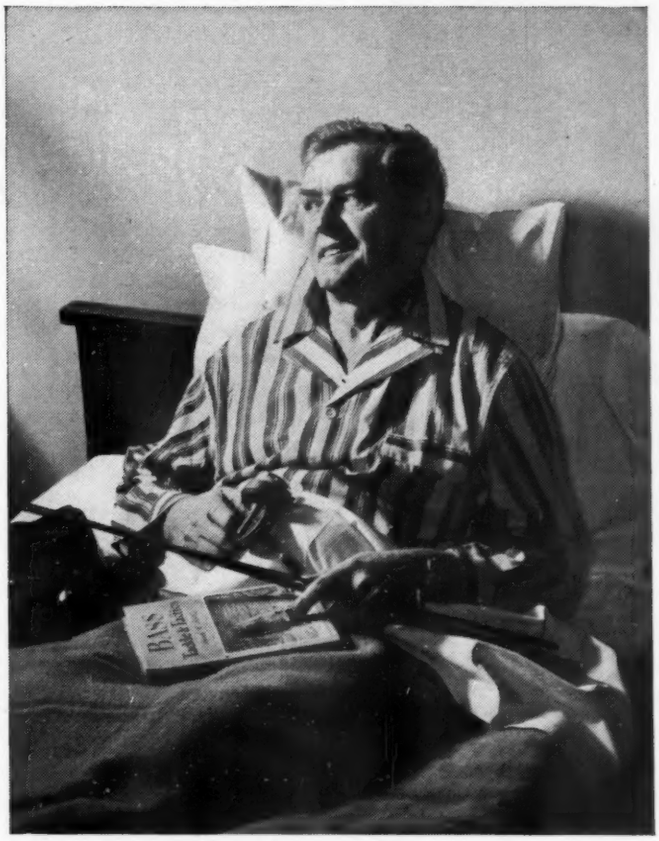

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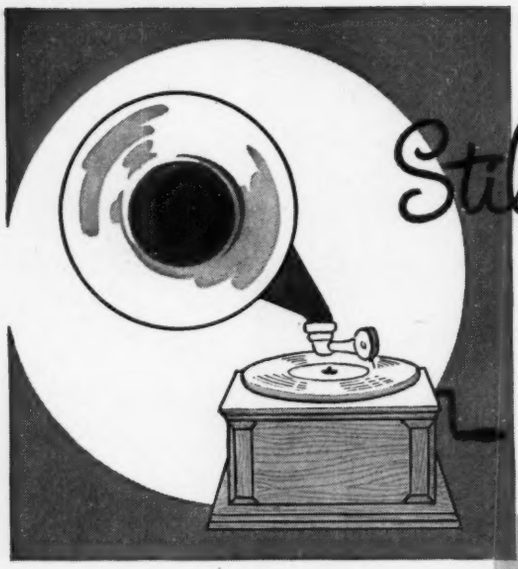

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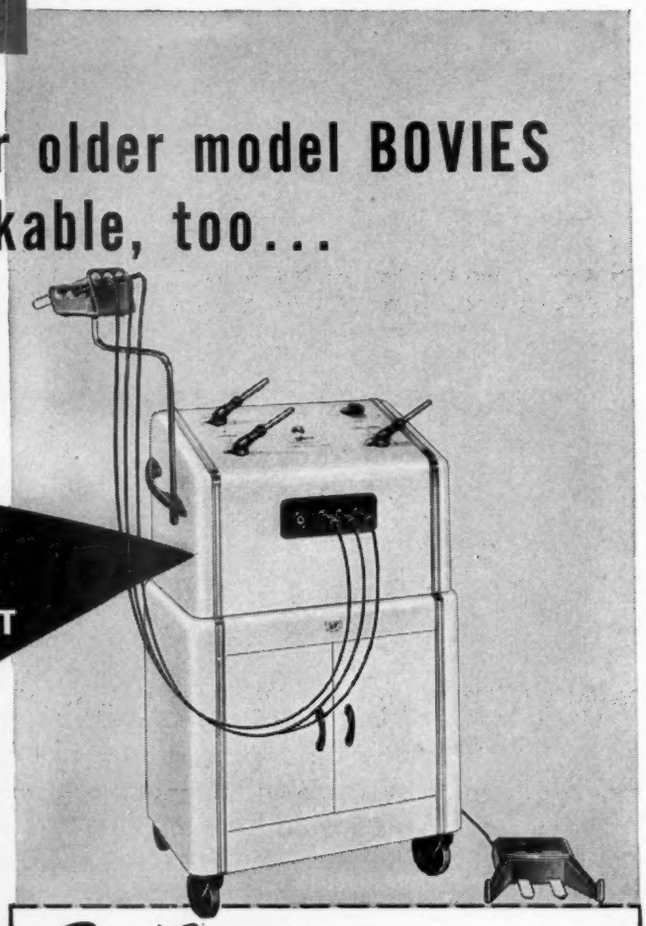

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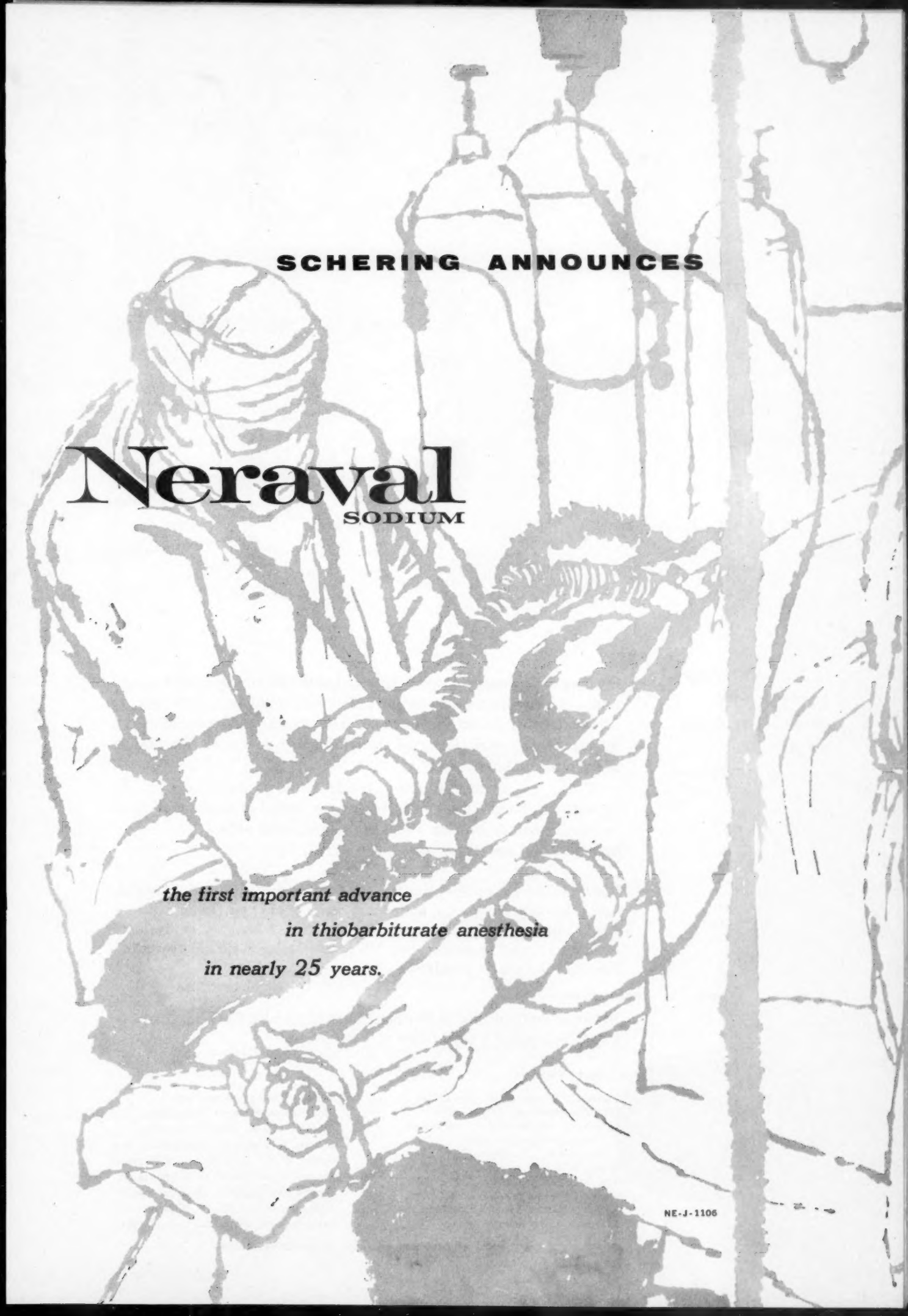

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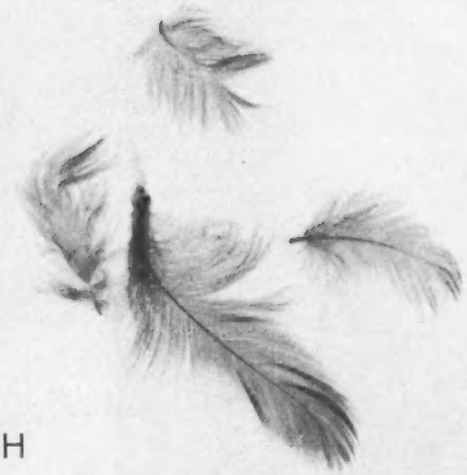

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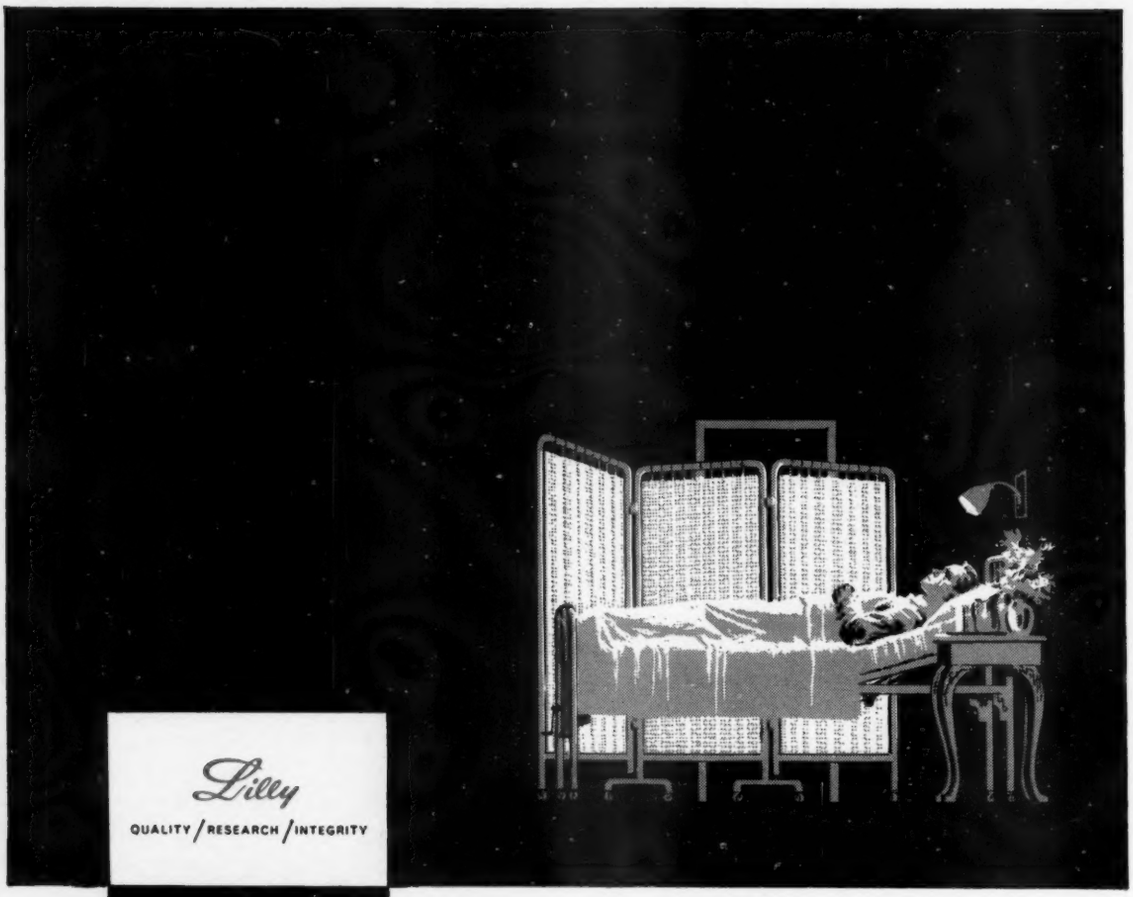

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The American Journal of Surgery

VOLUME 93 FEBRUARY 1956 NUMBER TWO

Foreword

WITHIN the past decade there have been numerous advances in the field of esophageal disease. Until recently our knowledge of this subject had been rather scant, and our diagnostic and therapeutic armamentarium had been limited.

Today, however, the picture is considerably brighter. Many patients have an excellent chance for cure while others are offered new hope for the improvement and prolongation of life.

In view of this it seemed timely to make available in one issue a compilation of present data relative to the diagnosis and treatment of diseases of the esophagus.

The contributors are leaders in their field and I am most grateful to them for making this issue possible.

EMIL A. NACLERIO, M.D.,
New York, New York

Newer Concepts of the Physiology of Deglutition and Dysphagia

NICHOLAS C. HIGHTOWER, JR., M.D., *Temple, Texas*

From the Department of Internal Medicine, Section on Gastroenterology, Scott and White Clinic, Temple, Texas.

THE act of swallowing is so taken for granted that little thought is given this highly complicated process unless some condition or disease state results in a disturbance of one or more of its integrated mechanisms. Within the past decade, however, interest has increased in the mechanisms involved in normal deglutition and in the disturbances of deglutition produced by various diseases. This heightened interest has occurred with the development of new technics for the study of esophageal motility.

Much of our original information concerning the act of swallowing resulted from roentgenologic observation of swallowed opaque media and the employment of balloon-tambour-kymograph methods of recording esophageal motility. Within the past few years, small sensitive pressure transducers have been developed and it is now possible to measure and record accurate and continuous tracings of esophageal intraluminal pressure [*1–3*]. All of these methods of studying deglutition have been refined and used alone or in combination to study the swallowing act in normal persons and in patients with dysphagia. Also, the effects of many pharmacologic agents on the normal and diseased esophagus have been determined [*4–6*]. The correlation of such investigations has evolved newer concepts of deglutition in health and disease.

NORMAL DEGLUTITION

Deglutition is divided into three stages. The first stage is voluntary, and the second and third stages are involuntary or reflex in origin. The first stage consists of the passage of food or liquid from the mouth into the pharynx. The second stage consists of the passage of the swallowed material through the pharynx. The third stage is concerned with the transport of the material through the esophagus and on into the stomach. After deglutition is initiated as a voluntary act, the second and third stages occur automatically and follow the first stage in rapid succession. The afferent limb of the reflex arc is principally the pharyngeal branches of the glossopharyngeal nerve. The efferent limbs of the reflex arc are the vagi. Central connections of the arc are thought to be in the medulla near the vagus nucleus.

First Stage. The first stage of deglutition is the voluntary act of forcing liquid or a bolus of food into the pharynx. In the case of solid food, the act is usually preceded by mastication which serves to soften and reduce particle size and form a bolus by moistening and mixing the material with saliva. The formed bolus, which has been thus softened and lubricated for its passage through the pharynx and esophagus, next is maneuvered to the dorsum of the tongue. The tongue then is elevated against the hard palate and retracted. This action, which is produced principally by the mylohyoid, styloglossus and hypoglossus muscles, forces the liquid or bolus into the pharynx.

Second Stage. When the material from the mouth comes in contact with pharyngeal and peripharyngeal structures, reflexes are initiated which accomplish the completion of the second

and third stages of deglutition. The base of the tongue, tonsils, anterior and posterior pillars of the fauces, soft palate, uvula and the posterior pharyngeal wall are all sensitive areas which, when stimulated by tactile stimuli, will initiate the swallowing reflexes. The principal nervous pathway is the glossopharyngeal nerve. Cocainization of the aforementioned sensitive areas inhibits the swallowing reflexes.

In order for the swallowed material to continue on its intended course into the esophagus, it is necessary that other pathways into the pharynx be closed. This is accomplished by a number of almost simultaneous actions. The swallowed material is prevented from reentering the mouth by the tongue which has remained retracted and elevated against the hard palate and, also, by the posterior pillars which contract and tend to close off the cavity of the mouth. Above, the nasopharynx is sealed off by the elevation of the soft palate as it comes in contact with the posterior pharyngeal wall. The entrance into the larynx and trachea is effectively closed off by the elevation and forward movement of the larynx, due mainly to the action of the anterior portions of the suprahyoid musculature. This results in the larynx coming to rest under the base of the tongue and, also, opens up the pharynx anteriorly. Both the true and false vocal cords are approximated and this further aids in sealing off the respiratory pathway.

At the completion of the previously described events, which requires only a fraction of a second, the pharyngeal constrictors contract to produce a rapid peristaltic wave. This wave arises at about the level of the laryngeal opening and propels the bolus toward the esophagus. The cricopharyngeus muscle, which has kept the esophagus closed until now, relaxes as the oncoming bolus approaches and allows it to enter the upper esophagus. The peristaltic contraction continues into the esophagus to become the primary esophageal peristaltic wave.

Third Stage. As in other parts of the alimentary canal, transport within the esophagus is dependent upon a gradient of pressure to move material within the lumen. The principal factor producing this pressure gradient is the primary esophageal peristaltic contraction which originates in the pharynx and is passed on into the esophagus. In transport of a solid bolus, the primary peristaltic contraction is of particular importance. It sweeps the bolus ahead of it as it progresses down the esophagus. When, due to size, consistency or dryness of the bolus, the primary peristaltic wave is ineffective in completing the transport of the bolus through the esophagus, secondary peristaltic contractions occur which complete the transport of the bolus. The latter contractions, produced by bolus distention of the esophagus, are initiated locally just above the bolus.

The primary peristaltic contractions, as observed fluoroscopically, have been described in considerable detail by Templeton [7]. The peristaltic contraction travels from its origin in the pharynx down the esophagus in an unbroken manner to the level of the diaphragm. The rate of propagation of the peristaltic waves becomes progressively decreased as it descends the esophagus. The fastest rate of travel in the upper esophagus is thought to be due to the striated muscle found there [8]. Butin and associates [9], by actual measurement of the time required for the primary peristaltic wave to reach different levels of the esophagus, found that the peak pressure produced by the wave occurred in the upper esophagus in 3.2 seconds, in the middle esophagus in 6.3 seconds and in the lower esophagus in 9.7 seconds. From data such as these, it has been computed that the velocity of a primary peristaltic wave varies from 2 to 3 cm. per second [3,9].

As the primary peristaltic contraction passes down the esophagus it produces a characteristic pressure pattern which can be recorded and which has been described in detail [3,9]. The pressure pattern consists of an initial negative wave followed by three positive waves. The most prominent component of the deglutition pressure pattern, as recorded from the mid- and lower esophagus, is the final large, simple, monophasic, positive pressure wave which rises rapidly to a peak pressure and declines with equal speed. The amplitude of this pressure wave is usually from 75 to 100 cm. of water pressure. Its duration is from two to three seconds [9,10].

The secondary peristaltic contraction produces a pressure wave similar to the final pressure wave of the deglutition pressure pattern. It is, also, a simple, monophasic, positive pressure wave. Pressure waves due to secondary peristaltic contractions produced in response to local distention by a small balloon have been recorded and measured [2]. They were observed

to occur at a rate of four to six per minute. The mean peak pressure was found to be approximately 30 cm. of water, and the duration of the waves seven to eight seconds.

The primary and secondary peristaltic contractions recently have been studied experimentally in the dog by Hwang [*11*]. Many of the observations reported are similar to those found in man.

The manner in which liquids are transported through the esophagus is dependent upon the position of the subject since gravity plays an important role. In the upright position, liquids travel through the esophagus at a speed that greatly exceeds the rate of propagation of the primary peristaltic wave. This rapid transport of liquids to the lower end of the esophagus is considered to be due to propelling force created by the contraction of the pharyngeal constrictors during the second stage of deglutition, as well as by the action of gravity upon the swallowed liquid. In the pharynx, near the end of the second stage of deglutition, the intrapharyngeal pressure is mounting due to contraction of the pharyngeal constrictors. The cricopharyngeus suddenly relaxes and opens the upper end of the esophagus which has a negative intraluminal pressure. The liquid is then more or less squirted into the lumen of the esophagus. In the upright position, the rapid passage of liquids through the esophagus requires only a few tenths of a second, and occurs while the esophagus is still relaxed and before the primary esophageal peristaltic contraction has begun. Fluoroscopy demonstrates that swallowed liquids are usually arrested at the lower end of the esophagus and await the arrival of the peristaltic contraction before they are admitted into the stomach. At times, however, when a series of swallows occur rapidly, as when drinking a glass of water, the liquid may pass on through the cardia with little pause and enter the stomach within a second or two after drinking has begun. When a quick succession of swallows occurs, the primary peristaltic wave is inhibited until the last swallow is finished. The peristaltic wave then passes down the esophagus emptying any remaining contents. The force of gravity is eliminated in either the supine or head-down position, and under these conditions liquid is handled in a manner similar to a solid bolus and transport is dependent upon the primary peristaltic wave.

The exact mechanism of esophageal evacuation into the stomach has been a matter of much dispute and still has not been established with certainty. Lerche [*12*] has postulated a mechanism of esophageal evacuation which is based on detailed anatomic studies and roentgenographic correlations. He has described two dilatations at the lower end of the esophagus. The upper one is called the esophageal ampulla and the lower one the gastroesophageal vestibule. The ampulla is simply a bulbar dilatation of the lower end of the esophagus. It is separated from the vestibule by the "inferior esophageal sphincter." The gastroesophageal vestibule is separated from the stomach by the "constrictor cardiae" which also is said to have sphincteric action. The intradiaphragmatic portion of the esophagus is the gastroesophageal vestibule. The diaphragm encircles the vestibule at a level of about 1 cm. below the inferior esophageal sphincter.

Lerche believes the inferior esophageal sphincter normally remains tonically closed and opens mechanically or reflexly to peristaltic pressure above the ampulla. After the inferior esophageal sphincter has opened, the ampulla contracts forcing its contents into the vestibule. When the vestibule is filled, being closed below by the constrictor cardiae, the inferior esophageal sphincter contracts. The constrictor cardiae now relaxes and the vestibule contracts and shortens itself, emptying its contents into the stomach.

Sanchez, Kramer and Ingelfinger [*3*] have reported pressure changes in the distal esophagus during deglutition. They adopted the anatomic terminology proposed by Lerche. In the esophageal ampulla, they recorded a pressure pattern following deglutition which was similar to that observed in the lower esophagus, but different in some important respects. The positive pressure wave due to the primary peristaltic contraction was observed to rise rapidly to a peak; but, instead of dropping off rapidly, it returned to the base line in a gradual slope. The pressure wave had a duration of fifteen to twenty-two seconds and traveled at a rate of 0.7 to 1.2 cm. per second. This evidence is compatible with fluoroscopic observations which show that the peristaltic wave slows down as it reaches the lower esophagus. Characteristic ampullary pressure waves were also recorded from a patient with a large hiatal hernia and from a patient with paralysis of the

left diaphragm, indicating the pressure pattern represented esophageal peristalsis.

From the vestibule, a decidedly different pressure pattern was recorded. Here only a gradual rise and fall of pressure lasting twenty to forty seconds was observed. The maximum pressure attained was only 5 to 15 mm. Hg. The type of pressure pattern recorded from the vestibule indicates that the vestibule is not in communication with the lower esophagus but is closed off, possibly by the inferior esophageal sphincter as suggested by Lerche. This is in keeping with the fluoroscopic observation that liquids frequently collect in the lower esophagus above the diaphragm and await the arrival of a peristaltic wave before being emptied into the stomach. These pressure observations in the distal esophagus seem to support the mechanism of esophageal evacuation proposed by Lerche. They would not support the mechanism of esophageal emptying proposed by Mosher [13] in which the diaphragm supposedly takes an active part.

In a recent roentgenologic study of the lower esophagus and esophagogastric junction by Poppel and associates [14], the findings confirmed Lerche's anatomic concepts of the ampulla, vestibule, inferior esophageal sphincter and the constrictor cardiae at the esophagogastric junction.

DYSPHAGIA

Dysphagia is a symptom produced by difficulty in swallowing. Thus, a disturbance of any one or more of the stages of deglutition may give rise to this symptom. Pain may or may not be associated. A great variety of diseases, including some of psychic origin, may result in dysphagia. The occurrence of dysphagia may indicate serious disease and should not be considered lightly. Usually, the presence of this symptom warrants thorough clinical investigation, including roentgenoscopic and endoscopic examination of the esophagus. When dysphagia is present for prolonged periods or when it is of a progressive nature, it is almost always associated with a marked loss of weight. For purposes of discussion, conditions producing dysphagia will be considered in relation to the stage of deglutition involved.

DISTURBANCES OF DEGLUTITION BY DISEASE

First Stage. A number of diseased states, including inflammatory processes, neoplasms and congenital anomalies may produce dysphagia by interfering with the first stage of deglutition. Any painful inflammatory process involving the mouth or tongue may make swallowing difficult. Aphthous ulcerations or stomatitis, acute papular glossitis, abscess of the tongue, tonsillitis and formation of granulomas are examples of inflammatory processes that may produce dysphagia by interfering with the first stage of deglutition. Such conditions are usually associated with pain. Although relatively rare, neoplasms of the mouth and tongue may be the cause of dysphagia. These lesions usually interfere mechanically with the movements of the tongue that are essential to swallowing and thus produce dysphagia. Tumors of the tonsils may block the passageway into the pharynx. The two most common congenital anomalies that produce dysphagia by interfering with the first stage of deglutition are cleft palate and elongation or hypertrophy of the uvula. In cleft palate, the tongue forces food or liquid into the nasopharynx and in cases of elongation of the uvula, mechanical blockage may occur.

Second Stage. A number of infections, neoplastic and neurologic disease, both local and systemic, are capable of producing dysphagia by interfering with the second stage of deglutition. By far, the most common cause of dysphagia involving this stage is acute pharyngitis. Peritonsillar and retropharyngeal abscess, mumps, scarlet fever, diphtheria and acute thyroiditis are all associated with dysphagia. Fixation of the larynx by chronic inflammatory processes such as tuberculosis and syphilis may make swallowing extremely difficult. Neoplasms of the larynx or thyroid may also produce fixation of the larynx thus preventing its upward and forward movement as normally occurs during the second stage of deglutition. Fixation of the larynx produces dysphagia, for mobility of this structure is essential to the second stage of deglutition. Lymphoma of pharyngeal and tonsillar origin may mechanically block deglutition during the second stage.

Pharyngeal paralysis is a common cause of dysphagia. In such instances, it may be impossible for the soft palate to be elevated, thus allowing liquids to be regurgitated through the nose. The pharyngeal constrictors may be unable to initiate the peristaltic contraction and empty the contents of the pharynx into the

esophagus. Pharyngeal paralysis usually results from bulbar lesions as seen in poliomyelitis, multiple sclerosis, cerebrovascular hemorrhage and syringomyelia. Cranial nerve (glossopharyngeal) neuritis may also result in pharyngeal paralysis. Myasthenia gravis is capable of producing dysphagia during this stage of deglutition. Palatal and pharyngeal paralysis sometimes follows diphtheria. Pharyngeal paralysis due to bulbar lesions may imitate neoplasms of the esophagus [*15*].

The syndrome of dysphagia, glossitis, hypochromic anemia, and sometimes splenomegaly and achlorhydria has been a subject of some disagreement. This combination of disorders, known as the Plummer-Vinson syndrome, has been adequately described in the past [*16,17*]. The dysphagia that occurs in this syndrome has been thought to be psychic or hysterical in origin due to achalasia of the cricopharyngeus muscle [*18*] or due to a pharyngoesophagitis associated with the syndrome [*19*]. Bockus points out that the common denominator in all cases is an iron deficiency anemia. He believes the dysphagia is due to a pharyngoesophagitis and is merely a complication of a specific type of iron deficiency anemia, associated with a vitamin B complex deficiency.

The study of such cases by measurement of pharyngeal and esophageal intraluminal pressures and correlation of such observations with careful fluoroscopic studies should increase our understanding of the disordered motor activity in these patients.

Third Stage. There are many disorders that may interfere with the third stage of deglutition. Within recent years, we have come to understand a number of them much better as newer technics of measuring intraluminal pressure have been applied to the study of esophageal transport. The response of the diseased esophagus to the action of various pharmacologic agents that act on the autonomic nervous system also has given us a better comprehension of the pathophysiology of some of the diseased states that involve the esophagus.

ACHALASIA

Achalasia of the esophagus is characterized by dysphagia which usually becomes progressively worse but may be intermittent, particularly in its early stages. Patients first experience the sensation when swallowed material lodges in the lower esophagus, producing a vague discomfort or pressure feeling in the lower substernal area. More difficulty is experienced with solids than liquids, but very hot or cold liquids may offend as much as solids. As the disease progresses and the esophagus becomes dilated and elongated, regurgitation and pain usually occur.

The term achalasia was first proposed by Hurst when he recommended its use instead of cardiospasm [*20*]. It generally is agreed today that achalasia is a disorder which involves most of the esophagus and does not represent "spasm of the cardia" [*21*]. Pathologic studies have repeatedly shown a degeneration of the myenteric plexus especially in the lower parts of the esophagus, and it is presumed that this represents the fundamental pathologic change or disorder [*22–25*]. What causes the degeneration of Auerbach's plexus is not known. This intrinsic nerve supply of the esophagus is probably concerned with local reflexes, and integration and propagation of peristaltic contractions.

The dysphagia in achalasia is due to a motor disturbance of the esophagus manifested mainly by an incomplete propagation of the primary peristaltic wave. There is also an apparent inhibition of reflex relaxation of the inferior esophageal sphincter. The primary peristaltic contraction originates in the pharynx in a normal manner, but when it has reached the level of the upper esophagus it seems to become ineffective, weaker and finally deteriorates and fades away. Secondary peristaltic contractions occur due to local distention by retained food and secretions, but once initiated these contractions move purposelessly up or down the esophagus for a short distance, then disappear. Without propagation of peristaltic contractions no intraluminal pressure gradient is created and esophageal transport is markedly retarded. As the normal motor functions progressively fail, retention of solid food, liquids and secretions occurs which leads to dilatation and elongation of the esophagus.

Esophageal intraluminal pressure studies in patients with achalasia have provided a better understanding of the motor disturbances that occur in this condition. It has been demonstrated that the primary peristaltic wave in these patients produces abnormal deglutition pressure patterns [*3,9*]. When a pressure gradient does occur, it is much less than that observed in the normal esophagus. Abnormal pressure waves due to secondary peristaltic

contractions are in addition frequently observed. In the terminal esophagus, it has been demonstrated that relaxation of the esophagogastric junction does not occur following deglutition [2,6]. These observations support the view that the inferior esophageal sphincter and the gastroesophageal vestibule are activated reflexly by the normally propagated peristaltic contractions. Thus, in the absence of normally propagated peristaltic contractions, the esophagogastric junction remains tonically closed.

The esophagus in patients with achalasia demonstrates a peculiar sensitivity to mecholyl® [4,10]. When administered subcutaneously or intramuscularly, mecholyl produces a marked and sustained contraction of the esophagus which may be observed fluoroscopically or recorded by balloon-kymograph or pressure-detecting technics. This marked sensitivity to mecholyl is probably due to the defunct intrinsic nerve plexuses, for the response obtained following this drug is in keeping with Cannon's law of denervation [27].

DIFFUSE SPASM

The clinical entity of diffuse spasm of the esophagus was first described by Moersch and Camp in 1934 [28]. Dysphagia due to diffuse spasm characteristically occurs intermittently and is associated with pain. The etiology of diffuse spasm is unknown, but it is thought that the localized intermittent spasm that occurs, usually in the lower esophagus, is reflex in origin as it frequently is associated with upper gastrointestinal disease. In patients with diffuse spasm, about equal difficulty is experienced with swallowing either liquids or solids. Moersch and Camp have described three types of abnormal motor activity in these patients as observed fluoroscopically: there is diffuse irregular spasm of the lower half or third of the esophagus, diffuse narrowing of the lower esophagus suggesting tetanic spasm and multiple spastic segments of concentric narrowing. A characteristic of diffuse spasm is an absence of marked dilatation above the area of constriction.

The irregular and abnormal contractions observed in the lower esophagus appear to be independent of normal deglutition motor activity. It has been recently reported that these abnormal contractions produce pressure patterns that greatly exceed the amplitude and duration of that normally observed after deglutition [29]. It has been previously reported that in two patients with diffuse spasm the pressure waves produced by swallowing solids and liquids were reduced in amplitude [10]. More observations of this type are needed for a better understanding of the abnormal motor activity that occurs in diffuse spasm of the esophagus. Mecholyl does not produce a significant increase in esophageal intraluminal pressure in patients with diffuse spasm.

SCLERODERMA

Scleroderma is an example of a systemic disease that may produce dysphagia due to interference with the third stage of deglutition. The primary disorder of esophageal motility is the defective propagation of the primary peristaltic contraction. Also, there is a rather marked loss of muscular tone in the esophageal wall. As in achalasia, scleroderma involves most of the esophagus and is not confined to any particular segment, although the changes are usually more evident in the lower esophagus. The dysphagia that results from scleroderma usually is progressive, but may remain static for many months.

The primary peristaltic contraction originates normally in the pharynx and passes on into the esophagus, but the contraction fades out usually in the upper levels of the esophagus. Lindsay and associates [30] made fluoroscopic studies in five patients with scleroderma and found the primary peristaltic contraction usually progressed only to the level of the suprasternal notch. In one of their patients the primary peristaltic contractions did progress further down the esophagus but were too shallow to push the bolus along. Furthermore, the contraction differed from a normal peristaltic wave in that the esophagus behind the advancing wave did not remain contracted, but distended immediately.

Kramer and Ingelfinger [31] have studied four patients with scleroderma with a balloon-kymographic technic. They found a marked decrease in esophageal tone and noted that propulsion of the inflated balloon down the esophagus was slow or absent. The wave pattern recorded by the balloon from different levels of the esophagus was markedly diminished or absent.

In one patient with scleroderma in whom esophageal intraluminal pressures were meas-

ured, it was found that few deglutition pressure complexes reached the lower esophagus, and those that did were of very low amplitude [*10*]. After swallowing liquid or a solid bolus, a pressure gradient of only about 10 cm. of water was produced in the lower esophagus.

Mecholyl does not increase esophageal intraluminal pressure in patients with scleroderma, as is observed in patients with achalasia.

HIATUS HERNIA

Hiatus hernia is a common disorder that may produce dysphagia, particularly when associated with esophagitis and/or esophageal peptic ulcer. When hiatus hernia is complicated by esophagitis or peptic ulceration, pain is common and usually is referred to the xyphoid area, but may extend into the chest, neck or back. It may simulate the pain pattern of angina pectoris.

Hiatus hernia interferes with the normal competence of the esophagogastric junction by frequently allowing reflux of gastric content into the lower esophagus. Flood and associates [*32*] have demonstrated fluoroscopic evidence of reflux in eighteen of thirty-four patients with hiatus hernia. In a review of recent literature, Feldman [*33*] found that in 432 collected cases of esophagitis and/or esophageal reflux, 51 per cent were associated with hiatus hernia.

Lorber and Shay [*6*] have studied esophageal transport in patients with hiatus hernia fluoroscopically and have described a "dysrhythmia" of esophageal motor activity. They found a delay in esophageal transport as evidenced by an increase in esophageal emptying time. In the patients with delayed emptying time there was an apparent inability of the peristaltic waves to promote complete evacuation. Reflux from the lower to the mid- or upper esophagus usually occurred and was followed by irregular and disorganized waves.

MECHANICAL OBSTRUCTION

Dysphagia produced by mechanical obstruction of the esophagus may be caused by intrinsic or extrinsic lesions. Intrinsic lesions include congenital anomalies such as stenosis and bands; inflammatory processes, as in esophagitis due to peptic ulceration, or swallowed caustics which may result in stricture; neoplasms such as carcinoma and benign tumors; and foreign bodies. Examples of extrinsic lesions which produce mechanical obstruction are large inflammatory periesophageal lymph nodes, lymphomas and aneurysms.

In general, most conditions causing mechanical obstruction of the esophagus produce similar disturbances in motility. When a small incomplete obstructive lesion involves the upper or middle esophagus, the primary peristaltic contraction may be observed fluoroscopically to "skip over" the lesion. When the obstruction exists in the lower esophagus, the primary peristaltic wave usually progresses to the site of the lesion without interruption. In some instances, the waves will be shallow and not strong enough to force the barium past the stenosed portion. As most obstructive lesions occur in the lower esophagus, distention of the proximal esophagus with retention of secretions and swallowed material is not unusual. The distention acts as a stimulus for secondary peristaltic contractions, and the waves frequently are observed at the time of roentgenologic examination.

In two patients with mechanical obstruction, Kramer and Ingelfinger [*31*] found, with a balloon-kymograph technic, that there was a decrease in esophageal tone and that the wave pattern consisted of strong rhythmic contractions occurring at a rate of four to six per minute. In each patient the inflated balloon was carried down to the point of obstruction before its progress was arrested.

Deglutition pressures have been determined in one patient with a benign stricture of the distal esophagus [*10*]. Liquid produced a peak gradient of pressure of 67 cm. of water, whereas a solid bolus resulted in a peak pressure of 34 cm. of water. Although these pressures are not as great as those recorded from normal individuals, they exceed the deglutition pressures observed in achalasia, diffuse spasm and scleroderma. Mecholyl did not produce a marked increase in intraluminal pressure.

PSYCHIC DISTURBANCES OF DEGLUTITION

The gastrointestinal tract of man is probably more capable than any other organ system of reflecting the emotional state of a person or of responding to the life stresses to which he may be subjected. Although the mechanisms involved are little understood, life stress has an intimate relationship to gastrointestinal symptoms and disease and may influence the clinical

course of a great many gastrointestinal disorders [*34*]. The esophagus is no exception. The association of psychic disturbances in the Plummer-Vinson syndrome, achalasia and even hiatus hernia is well recognized.

Globus hystericus is a classic example of a psychic or emotional disorder producing dysphagia. These patients, who are usually suffering from great anxiety, complain of a "lump in the throat" that they are unable to swallow or regurgitate. Bockus [*19*] states, however, that patients with globus hystericus can usually swallow food without difficulty. The symptom disappears when the anxiety, grief or other emotional disturbance subsides.

Wolf and Almy [*35*] have shown that stressful stimuli are capable of prolonging esophageal emptying time in normal individuals as well as patients with achalasia. In many of the patients with achalasia, the discussion of emotionally charged topics produces a prompt and sustained decrease in esophageal emptying as observed fluoroscopically.

Thus, there is ample evidence that psychic influences can and probably frequently do influence the normal mechanics of deglutition; however, more investigation is needed to better understand the interesting relationship of psychic influences to deglutition.

SUMMARY

The development of technics for the accurate measurement and recording of esophageal intraluminal pressure coupled with careful roentgenographic observation have advanced our knowledge of the mechanical factors involved in deglutition and dysphagia. Transport within the esophagus is dependent upon intact mechanisms which are capable of creating a pressure gradient. A definite and characteristic normal deglutition pressure pattern is recognized. Abnormal pressure patterns as a result of local or systemic disease may produce a loss of pressure gradient and result in dysphagia. The exact mechanism of esophageal evacuation is still somewhat in doubt and further study is needed to elucidate the action of the distal end of the esophagus. The esophagogastric junction is a delicately integrated structure and disturbances may produce a functional obstruction or allow esophageal reflux.

A number of mechanical, inflammatory, neoplastic, neurologic and psychic disorders may produce dysphagia by interfering with one or more of the stages of deglutition. Dysphagia may indicate a serious condition; therefore, its presence usually warrants a thorough investigation, including roentgenographic and endoscopic examination of the pharynx and esophagus.

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Roentgen Differential Diagnosis of Lesions Affecting the Esophagus

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Not only is the esophagus the site of a number of intrinsic disease processes but also, by its strategic location in the mediastinum, it serves as a reliable indicator of pathologic conditions in nearby structures.

Before considering the roentgen signs associated with abnormal conditions of the esophagus and their differential diagnosis, it may be fruitful to review briefly some of the problems encountered in the radiologic study of this organ.

In addition to the translation of a two-dimensional image into the morphology of a three-dimensional structure, roentgen study of the esophagus, like that of other parts of the gastrointestinal tract, entails demonstration and interpretation of its inadequately understood functional motor activity, which present certain difficulties. These are most troublesome at the poorly defined pharyngoesophageal and esophagogastric zones. Other problems of motor activity also encountered are those in relation to the inherent characteristics of all smooth muscles, that is, muscle tonus, peristalsis and other neuromuscular mechanisms under the influence of nervous, hormonal and pharmacologic stimuli.

The poorly defined zones mentioned are the subject of much speculation from an anatomic point of view [*27*]. There is no consensus [*9*] as to the morphology and physiology of the esophagogastric zone and its exact relationship to the diaphragmatic esophageal hiatus. Hence diversified opinions exist as to the definition of such terms as cardia [*16*], cardiac antrum, cardiac sphincter, phrenic ampulla, epiphrenic bell, inferior esophageal sphincter, gastroesophageal vestibule and abdominal portion of the esophagus.

Since a physiologic phenomenon is not, *ipso facto*, associated with an anatomic counterpart, artificially forcing such a premature correlation causes unnecessary confusion. From the roentgenologic point of view there is no question that some degree of sphincteric and relaxing activity occurs at the lower aspect of the esophagus [*28*]; but to assign a definitive anatomic site or structure to this physiologic zone leads to dogmatic interpretations which are not "operational"* since they cannot be proved or disproved. One such interpretation is the frequently made diagnosis of minuscule esophageal hiatal hernia of the stomach.

In x-ray examination of the esophagus with contrast media a cast of the lumen is visualized. Therefore any lesion of the mucosal surface or esophageal wall will produce, if sufficiently large, an abnormality of this luminal cast. The actual lesion itself is not seen, but only the effect of that portion of it which changes the contour of the barium column. It is generally accepted that any malignant lesion more than 5 cm. in length has almost invariably extended beyond the esophageal wall into the mediastinum, involving the lymph nodes, but this involvement of the mediastinum and lymph nodes usually is not demonstrable on x-ray. It is discovered only at operation or on postmortem study.

Roentgenologic examination of the esophagus entails, in addition to fluoroscopic and complete film study, certain other technical procedures or modifications as deemed necessary for a particular condition or situation.

The roentgen findings or signs listed in Table 1 will be used as a frame of reference rather than as criteria, since definite standards do not exist. It should be understood that some

* The term "operational" is here used in the sense expressed by P. W. Bridgmann.

TABLE I

ROENTGEN SIGNS IN LESIONS AFFECTING THE ESOPHAGUS

I. Silbouette, Position and Relief Pattern

CALIBER:	Narrowing	Dilatation
PROTRUSION:	Intrusion (filling defect)	Extrusion (pouch, channel or niche)
MARGIN:	Irregularity	Smoothness
POSITION:	Fixation	Hypermobility, displacement
LENGTH:	Shortening	Elongation
RELIEF PATTERN:	Disordered pattern	Absent pattern

II. Functional Motor Activity

TRANSIT TIME:	Acceleration	Delay, obstruction
PERISTALSIS:	Hyperperistalsis	Hypo- or absent peristalsis
OTHER MOTOR ACTIVITY:	Tertiary waves	Other disorders of motor activity
MURAL FLEXIBILITY:	Rigidity	Hyperdistensibility

III. Other Findings Common to Several Roentgen Signs

1. Length of esophageal involvement
2. Topographic distribution of lesion:
   (a) Pharyngoesophageal zone
   (b) Upper, middle and lower third of esophagus
   (c) Esophagogastric zone
3. Associated roentgen signs in adjacent and/or distant structures
4. Modification of signs by special procedures:
   (a) Physiologic maneuvers
   (b) Changes in position of patient
   (c) Special roentgen technical procedures
5. Roentgenographic evolution of the process: stationary, progressive or regressive

of these findings are of little concern in many conditions and that numerous lesions may reveal several of these signs with varying degrees of significance. In one projection a lesion may be manifested by one sign, whereas in another projection the same lesion in the same patient may be manifested by a different roentgen sign. For the purpose of our presentation the roentgen sign used is the one which seems most definitive, although the same lesion might be described by other terms. These roentgen signs, it may be noted in passing, are pertinent not only to the esophagus, but also to the entire gastrointestinal tract and to other tubulomuscular organs.

The "normal" in all of these findings may exist within a very narrow range or may include so wide a range as to make distinction between normal and abnormal not only difficult but also impossible. Marked deviations from normal are always obvious and frequently considered typical or "pathognomonic." However, it must be recognized that the magnitude of any single type of disease process has a spectrum-like depiction and that the small deviations as seen in early stages of a disease may be more important to the final well-being of the patient than those which are more extreme. The detection of cancer in its early phases, for example, is obviously more advantageous to the patient than the detection of this same cancer in its later, more advanced stages. The roentgen problems involved run parallel to, and encompass, those problems existing in the pathologic anatomy and physiology of the esophagus as well as those encountered in clinical medicine and endoscopic study of this organ.

ROENTGEN SIGNS: SILHOUETTE, POSITION AND RELIEF PATTERN

1. Shortening. Shortening of the esophagus may occur in the following ways:

Longitudinal muscle contraction of the esophageal wall is one mechanism of shortening since, as a tubulomuscular organ, the esophagus has the ability to contract longitudinally as well as in diameter. Therefore if the anchoring device at its lower end is lost or weakened, as is the case in esophageal hiatus hernia of the stomach or eventration of the left leaf of the diaphragm, adjustment of the longitudinal muscular elements takes place, resulting in most instances in a relative shortening of the esophagus.

Fibrotic contraction of the wall of the esophagus is another mechanism by which shortening

occurs. This may be a sequel to the corrosive action of ingested poisons or inflammatory processes such as peptic esophagitis or "reflux" esophagitis of long duration. It is most frequently observed in the lower third of the esophagus.

Extensive infiltration by scirrhous carcinoma into the wall of the esophagus may produce in rare cases shortening of the esophagus similar in type to that produced by fibrotic contraction. It is to be noted that in these two types the shortening of the esophagus causes the retraction of the esophagogastric junction superiorly through the esophageal hiatus, resulting in an esophageal hiatus hernia of the stomach.

Congenital anomalous development also accounts for some cases of shortened esophagus [*1,3*].

2. Elongation. Elongation may be seen in the following conditions:

Stretching as well as shortening is permitted by the tubulomuscular nature of the esophagus. For elongation to occur, in addition to the fixed attachment at its proximal end, the esophagus must be firmly attached also at its distal end. The esophagus having no separate adventitia from other mediastinal structures and its external coat being admixed with that of the aorta, any changes occurring in the aorta's configuration, such as elongation or tortuosity, produce a relatively similar displacement in the contiguous portion of the esophagus.

Decrease in tonus of the esophageal wall permits elongation to occur subject to internal pressure and stresses such as are present in achalasia.*

3. Narrowing. Narrowing may be concentric or eccentric and the segment involved may be relatively long or short. The latter may be single or multiple. Other characteristics found in these narrowings are: smooth edges, tapering, conical or funnel-shaped configuration, or irregularity of contour with serration, peaks or angulation. Narrowing of the lumen may also be caused by filling defects. The mechanisms which produce different types of narrowing are the following:

Compression or flattening of the esophagus results from extrinsic pressure produced by space-occupying lesions arising in, or adjacent to, the mediastinum. These include neoplasms, inflammatory masses, aneurysms, cysts, enlarged thyroid lobes or an enlarged atrium.

Intrinsic narrowing lesions of the esophagus may be caused by neoplastic, inflammatory, congenital or other miscellaneous conditions. Some examples of these conditions follow:

Neoplasms: Annular or infiltrating scirrhous carcinomas.

Inflammations: In the early stages local spasm produces narrowing; and if the inflammatory process persists or progresses, this leads to fibrosis as occurs following corrosive action of poisons, injuries resulting from swallowed or impacted foreign bodies, esophagitis of a peptic nature or reflux esophagitis.

Anomalous development: Stenosis, strictures or atresias.

Miscellaneous conditions: Web formation in Plummer-Vinson syndrome [*19*], and diaphragm formation protruding into the lumen (lower esophageal ring) [*21*].

"*Spastic conditions*" or muscular contractions are commonly seen without any visible gross organic lesion to account for the narrowing. However, pathologic changes in the intramural nerve cells and plexuses have been reported in cases of achalasia. Spasm also occurs in conjunction with local esophageal lesions associated with inflammation and foreign bodies, as previously mentioned.

4. Dilatation. This is similar to ectasia in all other tubulomuscular organs and is the result of one or more of the following:

Obstructive phenomena such as those occurring with many of the types of narrowing (stenoses) previously mentioned are frequently associated as well with dilatation above the constriction. There are, however, some cases of narrowing or relative stenosis which are not associated with dilatation of the esophagus above the lesion, indicating that the mechanism of the dilatation is not merely due to the constricting lesion. It must be related to the nature of the narrowing, the degree of constriction and the duration of the stenosis in addition to other factors as yet not well understood.

Atonicity or hypotonicity without obstruction is seen in conditions apparently due to neuromuscular dysfunction as occurs in myasthenia gravis or atony of old age.

* Synonyms for "achalasia" are many, including cardiospasm, idiopathic dilatation, neuropathic dilatation, phrenospasm, esophagectasia and megaesophagus, thus indicating the great diversity of opinions as to the etiology of this condition.

5. Intrusions or Filling Defects. Although these also cause narrowing, they are more conveniently treated as a separate sign. There is no sharp dividing line between certain of the cases of narrowing previously mentioned and some of those placed in this group of intrusions or filling defects. Individual cases are placed arbitrarily in one or the other group, depending on which seems more appropriate to the examiner. When radiopaque material is given, the lesion intrudes upon the barium column, producing a defect in the cast. The filling defect may be formed by:

Impacted foreign bodies such as food or fruit pits.

Pedunculated polyps [7].

Mucosal excrescences such as broad-based neoplasms or inflammatory masses.

Intramural extramucosal lesions including benign or malignant tumors, such as leiomyoma or leiomyosarcoma.

Extramural masses usually cause compression or constriction of the esophagus and come under the "narrowing" category, although occasionally they are more localized and manifest themselves as filling defects.

6. Extrusions. This group comprises lesions which project beyond the usual confines of the walls of the esophagus. Several types of extrusions are seen such as:

Pouch formation which is found in pulsion and traction types of diverticula. These usually occur singly but occasionally may be multiple.

Niche or crater formation results from erosions and penetration into or through the walls as occurs with peptic ulcer, neoplastic ulceration or other inflammatory ulcerations.

Channel formation may occur as a result of perforation of an esophageal lesion. This may have a direct extension into the tracheobronchial tree or pleural cavity, or the channel may extend into the mediastinum and have a blind end (sinus formation).

7. Margin Characteristics. Irregularity of margins may be found in the entire barium column as seen in some forms of chronic esophagitis, mediastinitis and extensive esophageal neoplasm. More frequently, the irregularity may be confined to the site of a localized lesion, as occurs in the irregular filling defects of neoplasms.

Smoothness of the margin may refer to the margins of a localized roentgen finding such as the smooth funnel narrowing of achalasia or smooth filling defect of a benign tumor. It may also refer to the entire barium cast of the lumen of the esophagus as seen in scleroderma.

8. Hypermobility and Fixation. Hypermobility is dependent upon change of position of the mediastinum as a whole occurring with different positions of the patient and with varying intrathoracic pressures. Fixation is the reverse, denoting immobility of the mediastinum as occurs in mediastinitis.

9. Displacement and Malposition. Pathologic changes in any of the surrounding structures may displace the esophagus, as is seen in left atrial enlargement, elongation and tortuosity of the aorta, large mediastinal masses, massive pulmonary atelectasis, massive pleural effusion, thoracoplasty and other thoracic and spinal deformities.

10. Relief Pattern Abnormality. Absence or smoothness of the mucosal folds may be of little or no significance since so often the folds depend on the state of filling of the esophagus with barium and the state of the muscularis mucosa. However, these "ironed out" folds become significant only when contiguous areas show normal folds, as occurs with extramucosal intramural lesions. Disordered mucosal folds such as coarseness, enlargement or changes in direction are encountered in varices, neoplastic involvement of the mucosa, "reflux" esophagitis and other inflammatory conditions.

ROENTGEN SIGNS: FUNCTIONAL (MOTOR) ACTIVITY

The following signs indicate changes in functional (motor) activity and are demonstrable by fluoroscopic and composite film studies. These signs are less tangible and more difficult of interpretation and evaluation than those discussed previously. They will probably assume greater importance and become easier to evaluate when cinematographic studies are more commonplace. A few institutions have carried out experimental kymographic studies to evaluate the functional motor activities of the esophagus [24] which have helped to elucidate the mechanisms of disordered motor activity.

1. Accelerated Transit. This finding is of little consequence radiologically. It is usually associated with irritability in inflammatory and corrosive lesions of the esophagus without the presence of obstructive phenomena.

TABLE II

ROENTGEN FINDINGS IN THE ESOPHAGUS IN A PILOT STUDY OF 100 CONSECUTIVE CASES*

Age Groups in Years	No. of Cases	No. of Abnormal Cases	Abnormal Roentgen Signs or Lesions (As Demonstrated on Films and Fluoroscopy)							Disordered Motor Function (As Observed Primarily by Fluoroscopy)			
			Carcinoma	Diverticulum	Esophagitis (Mild)	Displacement of Esophagus (Marked)	Hiatus Hernia	Problem Cases, Atypical Ampulla or Small Hiatus Hernia	"Schatzki Ring"	"Tertiary Waves"	Regurgitation	To-and-fro Movements	Other Neuromuscular Dysfunction
0–9	2	0	..	..	..	..	..	..	..	..	..	..	..
10–19	5	0	..	..	..	..	..	..	..	..	..	..	..
20–29	5	1	..	..	1	..	..	..	..	..	..	..	..
30–39	10	1	..	..	..	..	..	..	..	..	1	..	..
40–49	16	5	1	..	..	..	..	1	..	..	3	..	..
50–59	31	12	1	1	2	1	..	4	..	1	1	..	1
60–69	18	9	..	3	1	..	3	1	1	..	..	..	..
70 and over	13	11	1	..	..	..	1	3	..	4	4	1	..

* Careful fluoroscopic and film studies of the esophagus were carried out on 100 consecutive patients referred for x-ray studies for possible lesions in, or for complaints referable to, the gastrointestinal tract. All patients were studied in the erect, supine and Trendelenburg positions and subjected to the Valsalva, Johnstone and other maneuvers, except a few patients in whom these procedures were contraindicated.

2. *Delayed Transit and Obstruction.* Delayed transit of the barium or the complete stoppage of the flow of barium through the esophagus occurs in a number of conditions, as follows:

Obstructing, narrowing or constricting lesions of the esophagus, as mentioned previously.

Changes in the constituents of the wall such as occur with fibrotic replacement of muscle or carcinomatous infiltration.

Disordered neuromuscular mechanisms such as are seen in atony of old age.

3. *Peristalsis.* Peristaltic activity of the esophagus is integrated into the complex physiologic process of deglutition. This process is dependent upon the central nervous system, peripheral nervous system and the neuromuscular mechanisms within the esophageal wall. Therefore a derangement or abnormality, singly or in combination, in any of these three may result in disordered motor activity of the esophagus and interference with its normal peristaltic activity. The sphincteric action at both the pharyngoesophageal and esophagogastric zones are also coordinated with normal peristaltic activity, and consequently disturbance of peristaltic activity and/or sphincteric action may be deranged by the same mechanisms. In addition, abnormality in the function of one may adversely affect the other.

Alvarez speaks of reverse wavelets or ripples, but these are not considered true reverse peristalsis [5]. The so-called tertiary waves or "curling" effect of the esophagus is one of the manifestations of a deranged intrinsic neuromuscular mechanism frequently of little clinical significance and is not considered to be abnormal peristaltic activity but rather disordered motor function.

4. *Disordered Motor Function.* Neuromuscular dysfunctions of the esophagus are those conditions which interfere with the orderly, complex and coordinated muscular activity of the pharyngoesophageal zone, the esophagus and esophagogastric zones during the act of deglutition. These may be seen in such conditions as myasthenia gravis, scleroderma or diseases of the central nervous system. They also include that group of disordered movements of the esophagus as seen in such non-specific conditions as excessive regurgitation and to-and-fro movements of the barium in the esophagus, both of which are most frequently seen in patients in the older age group. (Table II.)

5. *Mural Flexibility.* The flexibility or lack of flexibility (rigidity) of the wall of the esophagus rarely occurs without the presence of other roentgen findings.

Increased distensibility may be seen either limited to a local segment or involving almost the entire esophagus. This may be of a transient nature, visible only on fluoroscopic study, or it may be on a more permanent basis with the formation of pseudodiverticula, as is occasionally seen in the aged. (Chart III.) In infants transitory neuromuscular dysfunction in the esophagogastric zone results in "chalasia" of

the cardia. This condition is an important cause of regurgitation of gastric contents into the esophagus resulting in severe and repeated vomiting [20].

Rigidity no doubt occurs; and although it cannot be demonstrated by ordinary technics, the cinematographic and kymographic technics show promise of revealing this condition in early lesions.

ROENTGEN SIGNS: OTHER FINDINGS COMMON TO SEVERAL SIGNS

1. Length of Esophageal Involvement. Many of the roentgen signs listed pertain to involvement of either long or short segments of the esophagus. For example, in corrosive esophagitis the resulting narrowing frequently involves a long segment, whereas in achalasia narrowing is almost always limited to a short segment.

2. Topographic Site of Lesions. The esophagus is conveniently divided into the pharyngoesophageal and esophagogastric zones at either extremity, and between these, the upper, middle and lower thirds. The location of the lesion may aid in the diagnosis since different diseases show a predilection for certain segments. For example, sideropenic anemia (Plummer-Vinson syndrome) almost invariably involves the pharyngoesophageal zone and/or the upper portion of the esophagus; myasthenia gravis, the upper third; traction diverticula, mostly the middle third; peptic esophagitis, the lower third; achalasia, the esophagogastric zone.

3. Associated Roentgen Findings in Adjacent and Distant Structures. Roentgen findings elsewhere may contribute to the evaluation of diseases of the esophagus. For example, the presence of mediastinal air may indicate esophageal rupture in the lower mediastinum and the presence of "aspiration pneumonitis" may lead to the suspicion of an esophageal lesion.

4. Modification of Roentgen Findings by Special Procedures. Physiologic maneuvers may provide additional information. For example, the Valsalva or a similar movement is frequently used to demonstrate the presence or absence of a small esophageal hiatal hernia of the stomach or as an aid in demonstrating the presence of esophageal varices.

Changes in the position of the patient as described by Templeton [27] and Johnstone [14] give various means of increasing the intra-abdominal pressure to demonstrate the presence of hiatus hernia and regurgitation. Johnstone advises full bending and coughing with the patient in the standing position. Another method makes use of the pressure changes which occur in the abdomen when the patient is placed in the prone position. Thus in the oblique projection contrast between the air-filled fundus of the stomach and possible hernia can be discerned. Other positions such as the Trendelenburg position and both lateral decubitus positions are also used by radiologists, and at times a lesion such as esophageal hiatus hernia or regurgitation may be demonstrated by one position or maneuver and not by others. The various technics may all have to be attempted in a particular case.

Table 11 gives the roentgen findings in the esophagus in a pilot study of 100 cases. With few exceptions (those in which the patient's condition did not permit) each patient was studied in the erect, supine and Trendelenburg positions and was subjected to the Valsalva, Johnstone and other maneuvers.

Special roentgen technical procedures such as barium-filled capsules or barium-impregnated bread or cotton are used in the study of swallowed foreign bodies impacted in the esophagus which are difficult or impossible to visualize due to their lack of radiopacity.

Lipiodol is better tolerated in the tracheobronchial tree than is barium. Therefore, in patients with suspected high esophageal obstruction with a tendency to aspirate, or in infants with swallowing difficulties, it is advisable to use lipiodol to demonstrate the type and site of the lesion.

Thick barium creams or pastes which have special adhering characteristics are used to demonstrate the mucosal relief pattern in such superficial lesions as mucosal ulcerations or esophageal varices.

5. Evolution of Esophageal Lesions as Demonstrated by Roentgenograms. As determined by follow-up studies or comparison with previous roentgenograms, the lesion is observed to be stationary, progressive or regressive. It is obvious that this knowledge is an aid in differential diagnosis.

ROENTGEN DIFFERENTIAL DIAGNOSIS OF ESOPHAGEAL LESIONS

Figures 1, 2 and 3 are composite diagrammatic sketches of representative cases which

are listed in corresponding Charts I, II and III. These charts show the roentgen signs and final diagnosis, location of lesions and the number of cases in each group. The cases include those from our own series and those selected from the literature [6,18,19,23,24,27]. The listing of the number of cases in each group is not intended for statistical evaluation or analysis, but merely to indicate an approximation of the relative frequency of each in the considered opinion of the authors. Certain of the conditions listed, however, such as the Plummer-Vinson syndrome and scleroderma, seem out of proportion to the actual frequency with which they are encountered in practice. Due to special interest in these relatively rare conditions and the unique problems they present, case reports and studies appear more often in the literature than their numbers warrant.

The following is a brief consideration of additional points in roentgen differential diagnosis of esophageal lesions.

It is obvious, but may bear emphasis, that similar signs are found in different lesions and that different signs may be found in the same type of lesion. Most of the roentgen signs occur in groups and are given in combined terms such as smooth narrowing, irregular narrowing, or a combination of narrowing with dilatation.

1. Narrowing or Stenotic Lesions. The roentgen finding of "narrowing," as demonstrated in Figures 1, 2 and 3 and their accompanying charts, is one of the major components in most esophageal lesions. This stenotic condition may or may not be associated with dilatation above the narrowing.

Narrowing with irregular margins in a long segment, as noted in Chart I, is carcinomatous in a large proportion of the cases. A few of these carcinomatous processes placed under "narrowing" could just as well have been placed under the category of "extensive irregular filling defects." It is important to note that some dilatation of the esophagus above the malignant lesion occurs not infrequently although the amount of dilatation is not conspicuous. (Fig. 4.)

Narrowing with smooth margins in a long segment is usually benign in nature and, as may be observed in Chart I, is somewhat more frequently than not associated with dilatation. In considering both the irregular and the smooth narrow lesions it is important to bear in mind that the actual narrowing or constricted portion of the esophagus may so decrease the amount of barium passing through the segment that the more distal portion of the esophagus, although normal, may also appear to be narrowed because of an insufficient amount of barium to dilate it to its normal width. Occasionally a special maneuver such as having the patient in the deep Trendelenburg position may reveal the inferior margin of the constrictive lesion.

Esophagitis in the lower third of the esophagus is apparently due to the reflux of gastric juice from the stomach into the lower end of the esophagus. Therefore terms such as reflux esophagitis and peptic esophagitis are similar in meaning. The clinical manifestation of esophagitis as well as the presence of esophagitis proved by endoscopic study may not be associated with any abnormal roentgen findings of the esophagus. If roentgen signs of blurred margins or abnormality of the mucosal folds and irritability are present, these findings may be considered evidence of esophagitis if positively correlated with the clinical findings [23]. Repeated bouts of reflux esophagitis may lead to stenosis or peptic ulceration in the lower third of the esophagus. Peptic ulcer is also considered by some authors to be secondary to the presence of ectopic gastric mucosa in the lower end of the esophagus. The resultant stenosis following esophagitis [4] with or without the presence of peptic ulcer, as noted in Charts I, II and III, may take several forms. It may be either a long or short narrow segment with irregular or smooth margins and may or may not be associated with dilatation above. These lesions may also result secondarily in a short esophagus and hiatus hernia [25].

Eccentric narrowing with smooth margins of a short segment is only occasionally seen in carcinoma. The web formation in Plummer-Vinson syndrome (idiopathic hyperchromic anemia or sideropenic anemia, a condition usually found in middle-aged women) is as a rule found in the upper end of the esophagus near the pharyngoesophageal zone and almost invariably on the anterior aspect.

Concentric narrowing or short segments of narrowing having smooth margins with little or no dilatation above are usually benign but occasionally such a lesion may be malignant. (Chart II.) A short narrow segment with irregular margins, with or without dilatation above, is found in about equal frequency associated

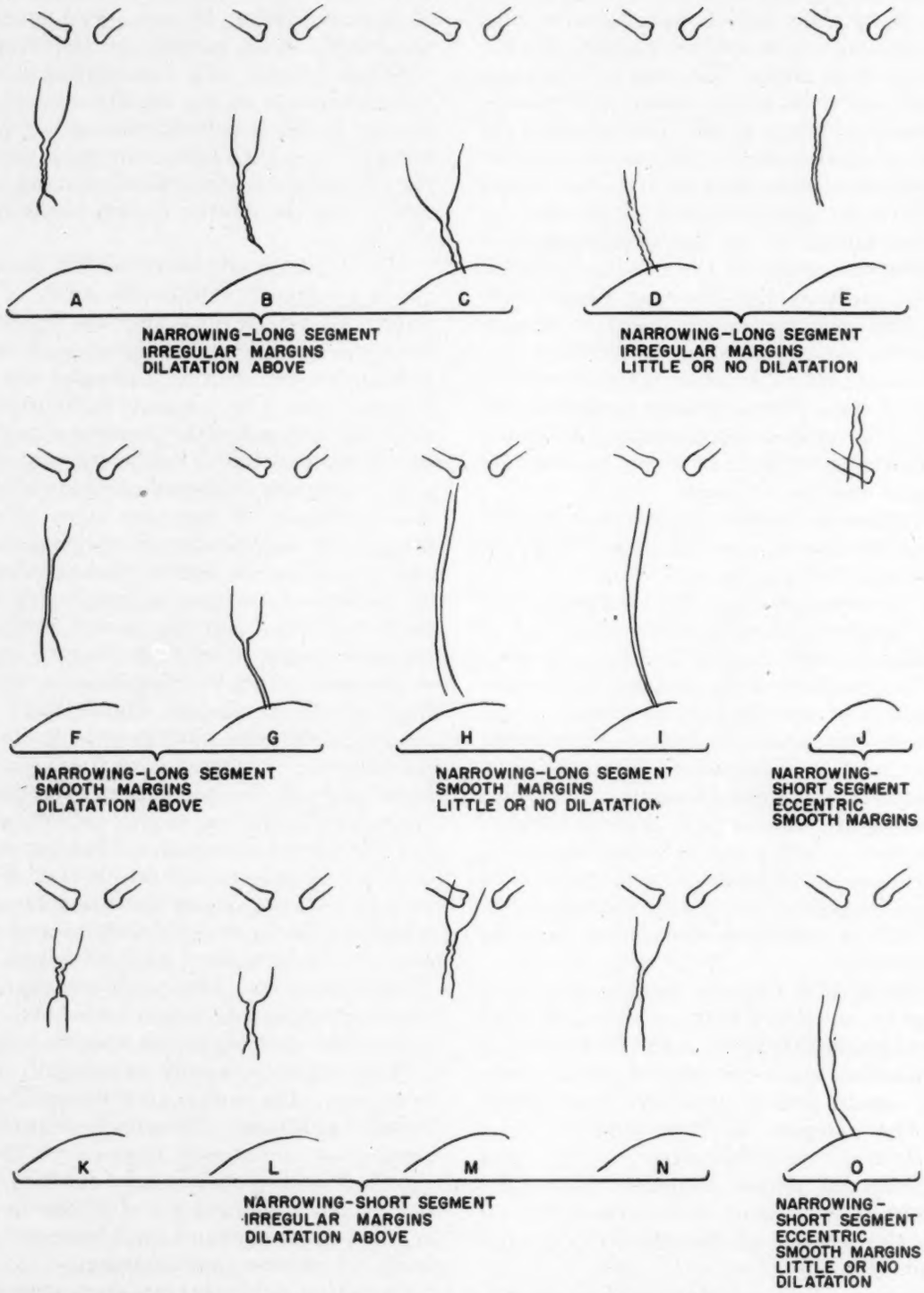

FIG. 1. Roentgen signs of esophageal lesions shown in composite sketches made from representative cases as given in Chart 1.

with cancer and benign stenotic lesions. (Charts 1 and 11.)

2. *Dilatation of the Esophagus.* When dilatation is marked and is associated with a smooth conical narrowing such as is seen in achalasia it is almost pathognomonic of a benign lesion. (Chart 11.) However, at times food particles or inflammatory areas at the site of the narrow-

Roentgen Differential Diagnosis of Esophageal Lesions

CHART I (SEE FIGURE 1)

LESION	NO. CASES	LOCATION
A, B, C. NARROWING - LONG SEGMENT IRREGULAR MARGINS DILATATION ABOVE		
CARCINOMA	7	U-2, M-2, L-3
STENOSIS (CORROSIVE)	4	M-1, M&L-2, L-1
STENOSIS (TUBERCULOUS MEDIASTINITIS AND GLANDS)	1	M
STENOSIS (METASTATIC NODES, CARCINOMA OF LUNG)	1	M
STRICTURE, BENIGN (CAUSE ?)	1	U
ESOPHAGITIS (REFLUX ?)	1	L

LESION	NO. CASES	LOCATION
D, E. NARROWING - LONG SEGMENT IRREGULAR MARGINS LITTLE OR NO DILATATION ABOVE		
ESOPHAGITIS	1	L
CARCINOMA	1	M
MULTIPLE STENOSIS WITH EROSION (CAUSE ?)	1	M&L

LESION	NO. CASES	LOCATION
F, G. NARROWING - LONG SEGMENT SMOOTH MARGINS DILATATION ABOVE		
STENOSIS (STRICTURE, BENIGN, CAUSE ?)	2	U-1, M & L-1
PULSION DIVERTICULUM	2	U
STENOSIS (CORROSIVE)	2	M, M & L
ESOPHAGEAL ULCER AND STENOSIS	1	L
OTHER INFLAMMATORY (E.G., SYPHILIS)	1	M

LESION	NO. CASES	LOCATION
H, I. NARROWING - LONG SEGMENT SMOOTH MARGINS LITTLE OR NO DILATATION ABOVE		
ESOPHAGITIS (PEPTIC)	2	L
STENOSIS (BENIGN, CAUSE ?)	1	L
SPASM	1	L
CONGENITAL NARROWING	1	M

LESION	NO. CASES	LOCATION
K, L, M, N. NARROWING - SHORT SEGMENT IRREGULAR MARGINS DILATATION ABOVE		
CARCINOMA	4	U-2, M-1, L-1
STENOSIS (BENIGN, CAUSE ?)	2	M
CARDIOSPASM WITH ULCER OR IRREGULARITY	2	L
PEPTIC ULCER AND STENOSIS	1	L
STENOSIS (TUBERCULOUS NODES)	1	M
STENOSIS (PLUMMER-VINSON SYNDROME)	1	U
SARCOIDOSIS	1	M
FIBROSARCOMA	1	M

LESION	NO. CASES	LOCATION
J, O. NARROWING - SHORT SEGMENT ECCENTRIC SMOOTH MARGINS LITTLE OR NO DILATATION ABOVE		
WEB (PLUMMER-VINSON SYNDROME)	3	U
SCIRRHOUS CARCINOMA	1	L

KEY TO LOCATION IN ESOPHAGUS

U – UPPER THIRD
M – MIDDLE THIRD
L – LOWER THIRD

ing may simulate a filling defect or changes that might be interpreted as a malignant process. (Chart 1, K, L, M, N.)

Smooth conical narrowing with some dilatation in the lower third of the esophagus is occasionally seen in the scirrhous type of carcinoma with submucosal infiltrations, as shown in Chart 11, F, G, H, and Figure 5.

These constricting carcinomatous lesions are usually located in the lower third of the esophagus (Fig. 5, A, B, D) and occasionally in the esophagogastric zone, where both the lower end of the esophagus and the fundus of the stomach may be involved. (Fig. 5, F.)

In this particular region the roentgenologic differentiation of this type of carcinoma from

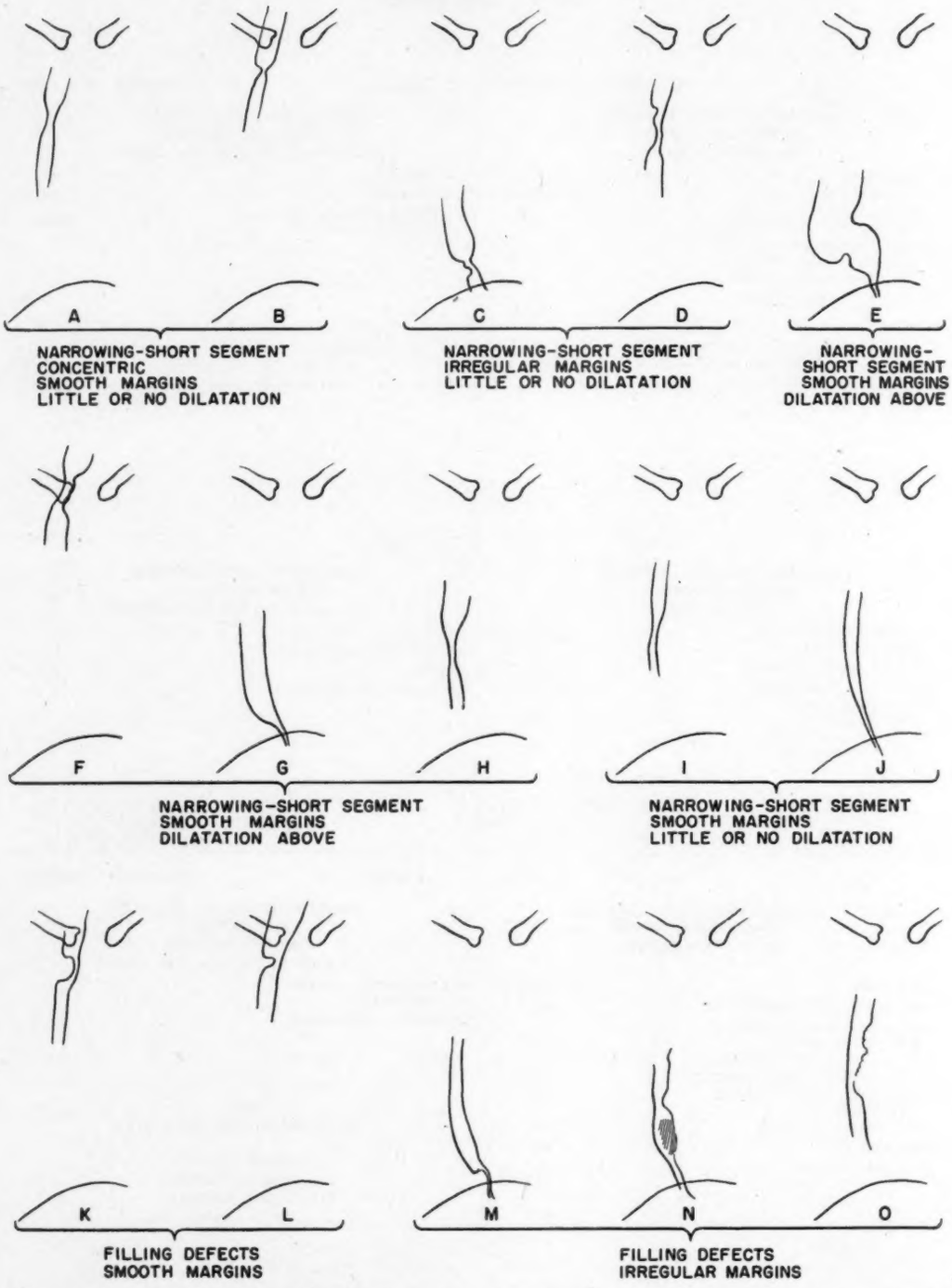

FIG. 2. Roentgen signs of esophageal lesions shown in composite sketches made from representative cases as given in Chart II.

some cases of achalasia or esophagitis may be difficult or impossible, and the high index of suspicion from a clinical point of view makes it essential that a suitable biopsy specimen be obtained by esophagoscopy.

Dilatation unassociated with a narrowing lesion is seen in benign conditions. (Chart III, J.) These conditions are as a rule diagnosed from the clinical history and findings. From a roentgenologic point of view, they are frequently associated with functional (motor) changes such as decrease or lack of peristalsis in sclero-

Roentgen Differential Diagnosis of Esophageal Lesions

CHART II (SEE FIGURE 2)

LESION	NO. CASES	LOCATION
A, B. NARROWING–SHORT SEGMENT CONCENTRIC SMOOTH MARGINS LITTLE OR NO DILATATION ABOVE		
STENOSIS (BENIGN, CAUSE ?)	4	M-3, L-1
ESOPHAGITIS, PEPTIC	3	L
CARCINOMA	2	U-1, M-1
STENOSIS (CORROSIVE)	1	L
CONGENITAL STENOSIS	1	M
CONSTRICTING BAND	1	U
"SCHATZKI RING"	1	L

LESION	NO. CASES	LOCATION
C, D. NARROWING–SHORT SEGMENT IRREGULAR MARGINS LITTLE OR NO DILATATION ABOVE		
CARCINOMA	4	M-3, L-1
STENOSIS (BENIGN, CAUSE ?)	1	M
ESOPHAGITIS	1	L
STENOSIS (TUBERCULOUS HILAR GLANDS)	1	M
SPASM (CAUSE ?)	1	L
POSTOPERATIVE DIVERTICULECTOMY	1	M
E. NARROWING–SHORT SEGMENT SMOOTH MARGINS DILATATION ABOVE (MARKED)		
ACHALASIA	8	L
SPASM (CAUSE ?)	2	L
INTRALUMINAL LARGE BENIGN TUMOR	1	L

LESION	NO CASES	LOCATION
F, G, H. NARROWING–SHORT SEGMENT SMOOTH MARGINS DILATATION ABOVE		
ACHALASIA	8	L
CARCINOMA	7	M-1, L-6
STENOSIS (CORROSIVE)	2	M-1, L-1
PLUMMER-VINSON SYNDROME	2	U
PEPTIC ESOPHAGITIS	1	L
SPASM	1	L
CARCINOMA, FUNDUS OF STOMACH, INVOLVING LOWER END OF ESOPHAGUS	1	L

LESION	NO. CASES	LOCATION
I, J. NARROWING–SHORT SEGMENT SMOOTH MARGINS LITTLE OR NO DILATATION ABOVE		
STENOSIS (CORROSIVE)	2	M-1, L-1
ESOPHAGITIS, STENOSIS	2	M-1, L-1
STENOSIS (BENIGN, CAUSE ?)	1	M
VERY EARLY ACHALASIA	1	L

LESION	NO. CASES	LOCATION
K, L. FILLING DEFECTS SMOOTH MARGINS		
PLUMMER-VINSON SYNDROME	3	U
FIXATION OF ENLARGED HILAR GLANDS	2	M
LEIOMYOMA	1	M
CARCINOMA	1	U
SPASM (CRICOPHARYNGEAL)	1	U

LESION	NO CASES	LOCATION
M, N, O. FILLING DEFECTS IRREGULAR MARGINS		
CARCINOMA	17	U-1, M-7, L-9
SARCOMA	2	M-1, L-1
FOREIGN BODY IMPACTION (FOOD)	2	L
BENIGN TUMOR	2	L
INFILTRATING MEDIASTINAL TUMOR	1	U

derma [11,17] and neuromuscular disorders which occur in atony of old age and secondary to changes in the central nervous system.

3. *Filling Defects. Filling defects with smooth margins* are usually benign and only rarely are they produced by a malignant process.

Filling defects with irregular margins are, with few exceptions, malignant. This is especially true when there is a tendency toward a shelf-like projection at the upper or lower end of the lesion. Occasionally benign lesions such as impacted food or fruit pits may be seen which simulate a malignant process. It is important to take cognizance of the fact that impaction of food may occur with an underlying benign or malignant stenotic lesion. All such cases

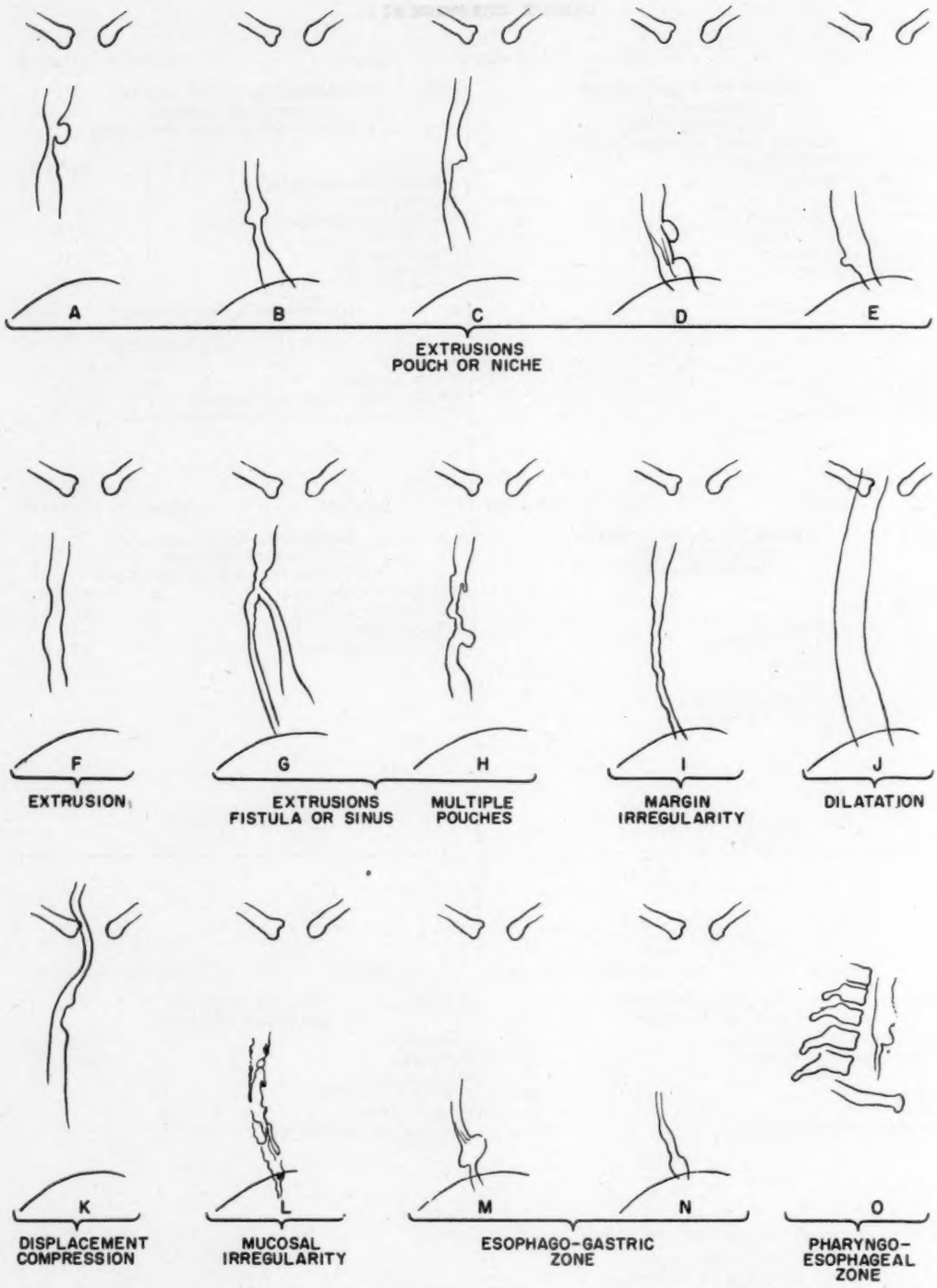

FIG. 3. Roentgen signs of esophageal lesions shown in composite sketches made from representative cases as given in Chart III.

should be adequately studied subsequent to the relief of the impaction to rule out a pre-existing lesion of the esophagus.

4. *Extrusions.* The two types of diverticula, traction and pulsion, are fairly clear-cut in typical cases as to location, size and configuration.

Traction diverticula are extremely common, Starck [26] claiming they are found in 5 to 6 per cent of all autopsies; however, most of these diverticula are of no clinical significance. They are usually located in the mid-third of the esophagus anteriorly, are small, have a

CHART III (SEE FIGURE 3)

LESION	NO. CASES	LOCATION
A,B,C. EXTRUSIONS - POUCH OR NICHE		
TRACTION DIVERTICULA	14	U-2, M-10, L-1
PULSION DIVERTICULA	10	U-4, M-2, L-4
ULCER NICHE.	2	M-1, L-1
CARCINOMA WITH PERFORATION	1	M

LESION	NO. CASES	LOCATION
D,E. EXTRUSIONS - NICHE OR POUCH		
PEPTIC ESOPHAGEAL ULCER.	3	L
LARGE MALIGNANT ULCER.	1	L
LARGE PULSION TRACTION DIVERTICULUM.	2	M

LESION	NO CASES	LOCATION
F. EXTRUSIONS - NICHE OR POUCH		
LARGE FLAT ULCER	1	L
TRACHEO-ESOPHAGEAL FISTULA, CHANNEL NOT VISIBLE	1	M
G. EXTRUSIONS - FISTULA OR SINUS		
CARCINOMA WITH ESOPHAGOTRACHEO-BRONCHIAL FISTULA.	5	M
TRACTION DIVERTICULUM WITH PERFORATION	2	M
CONGENITAL ESOPHAGEAL ATRESIA, ESOPHAGOTRACHEAL FISTULA	2	U
STENOSIS (CORROSIVE) WITH FISTULA	1	M
CONGENITAL DOUBLE ESOPHAGUS	1	M
H. EXTRUSIONS - MULTIPLE POUCHES		
MULTIPLE DIVERTICULA	4	M-2, L-2
PSEUDODIVERTICULA.	2	M-L
CORKSCREW ESOPHAGUS	2	M-L
SACCULATIONS SECONDARY TO CORROSIVE ACTION.	1	M
EXTENSIVE PROLIFERATIVE NEOPLASM	1	M

LESION	NO. CASES	LOCATION
I. MARGIN IRREGULARITY		
CHRONIC ESOPHAGITIS.	2	M-L
MULTIPLE FINE MALIGNANT ULCERATIONS.	2	M-L
LONG IRREGULAR SPASM	1	M-L
EXTENSIVE CARCINOMA.	1	L
POSTOPERATIVE ACHALASIA.	1	M-L
MULTIPLE INDENTATIONS FROM LARGE SPUR FORMATIONS OF SPINE.	1	U
"TERTIARY WAVES OR CURLING".	MANY	M-L
J. DILATATION		
ATONY OF ESOPHAGUS, CENTRAL NERVOUS SYSTEM SYPHILIS.	1	U-M-L
ATONY OF ESOPHAGUS (SENILE?).	1	M-L
SCLERODERMA	1	M-L

LESION	NO. CASES	LOCATION
K. DISPLACEMENT-COMPRESSION		
ELONGATION AND TORTUOUS AORTA	MANY	--
ANTERIOR DISPLACEMENT, RIGHT AORTIC ARCH.	3	U-M
LARGE LOBE OF THYROID	2	U
ABNORMAL COURSE OF ARTERY	1	U-M
LYMPHOSARCOMA OF MEDIASTINAL NODES	1	U-M
AORTIC ANEURYSM	2	U-M
METASTATIC MEDIASTINAL CARCINOMA OF LUNG.	1	M
L. MUCOSAL IRREGULARITY		
ESOPHAGEAL VARICES	MANY	L
CARCINOMA	3	M-L-1, L-2

LESION	NO. CASES
M,N. NORMAL FINDINGS AND LESIONS OF ESOPHAGO GASTRIC ZONE	
PHRENIC AMPULLA SIMULATING HIATAL HERNIA.	MANY
SMALL ESOPHAGEAL HIATAL HERNIA OF STOMACH.	MANY
SMALL HIATAL HERNIA AND PHRENIC AMPULLA.	SEVERAL
CARCINOMA, FUNDUS OF STOMACH AND LOWER END OF ESOPHAGUS.	4
ESOPHAGITIS (REFLUX?).	2
SCLERODERMA.	3
O. LESIONS OF THE PHARYNGO-ESOPHAGEAL ZONE	
LARGE INFILTRATING CARCINOMA OF HYPOPHARYNX AND ESOPHAGUS WITH SOME STENOSIS.	3
NEUROMUSCULAR DYSFUNCTION.	1

broad neck and at times do not fill well with contrast media. A small, horizontally peaked diverticulum found at the anterior aspect of the esophagus at the level of the hilar region of the lung is usually characteristic of the traction type of diverticulum.

Pulsion diverticula are found on the posterior wall in the region of the pharyngoesophageal zone and occur in persons in later life. The large diverticula of this type, known as Zenker's diverticula, usually develop to the left and show a fluid level with the patient in the erect position. The diverticulum may displace and compress the esophagus like a large unilateral thyroid tumor. The epiphrenic diverticula are usually of the pulsion type and are located

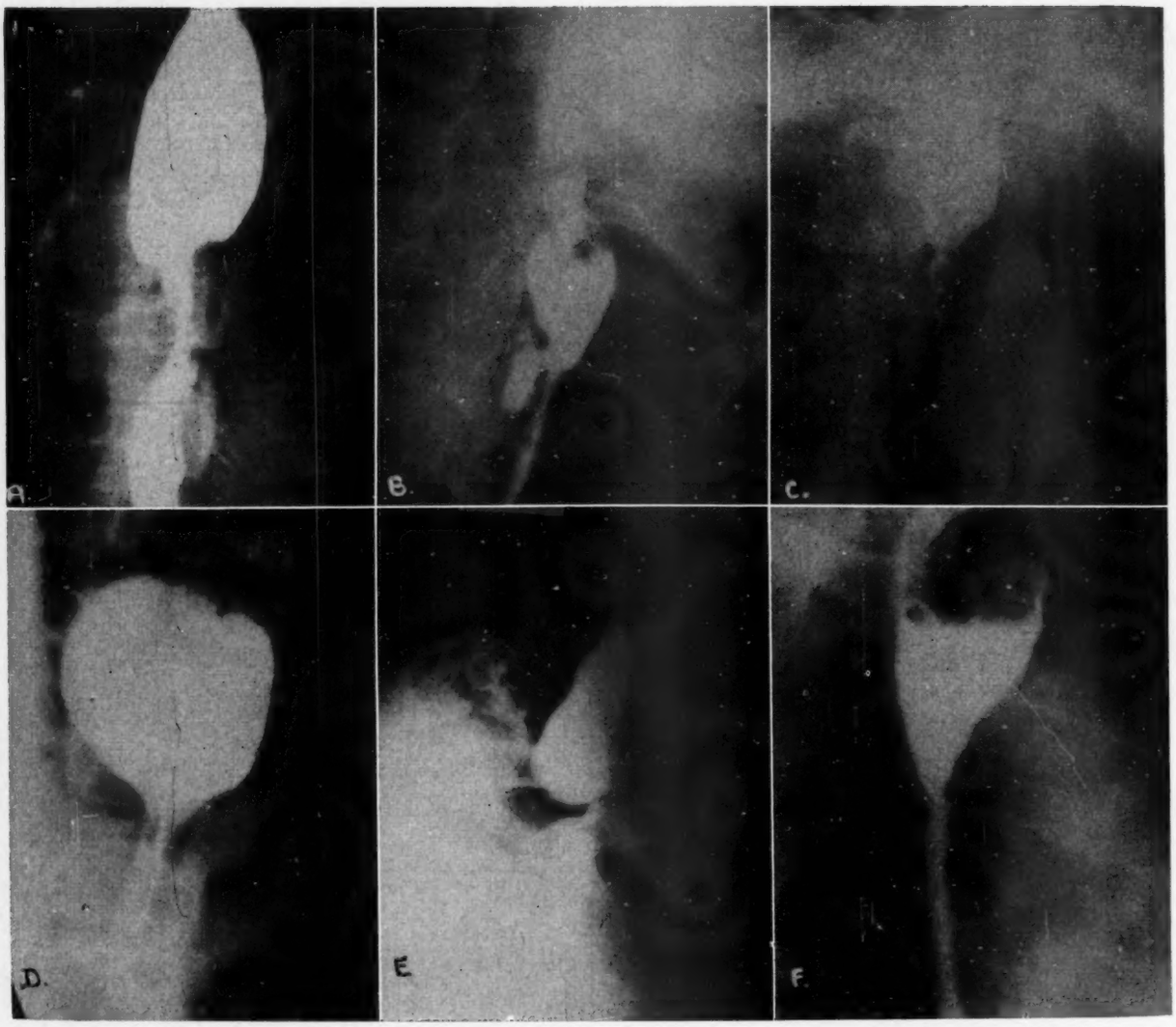

FIG. 4. Irregular narrowing with dilatation above: (A) carcinoma of the esophagus, (B) benign stenosis with diverticulum, (C) carcinoma of the esophagus. Smooth narrowing with dilatation above: (D) stricture (benign), (E) pulsion diverticulum, (F) corrosive stricture.

immediately above the diaphragm with dilatation of the esophagus above the lesion in many cases.

Combined forms of diverticula are also seen which apparently have both traction and pulsion characteristics. They may occur anywhere in the esophagus but are more frequent in the middle third. In this mixed type the descriptive findings as to the diverticulum's size, configuration, the direction of the lumen and the retention of barium or food are more important than an attempt at an etiologic diagnosis.

In some cases the diverticulum shows irregularity of contour and it may become inflamed, perforate and form a fistula. When this occurs, it makes the diverticulum difficult to differentiate from carcinoma although carcinoma is rarely found in a diverticulum.

Peptic ulcers of the esophagus have been partially discussed previously in connection with esophagitis. These ulcers are shallow as a rule, but occasionally may be large and simulate a diverticulum; rarely they may be flat, in which case it becomes difficult to differentiate them from other types of irregularity. This ulceration is usually located in the lower segment of the esophagus, frequently has some narrowing or stenosis associated with it, and there may be dilatation of the segment above this level. In many instances the small shallow ulcers are difficult to detect on x-ray although

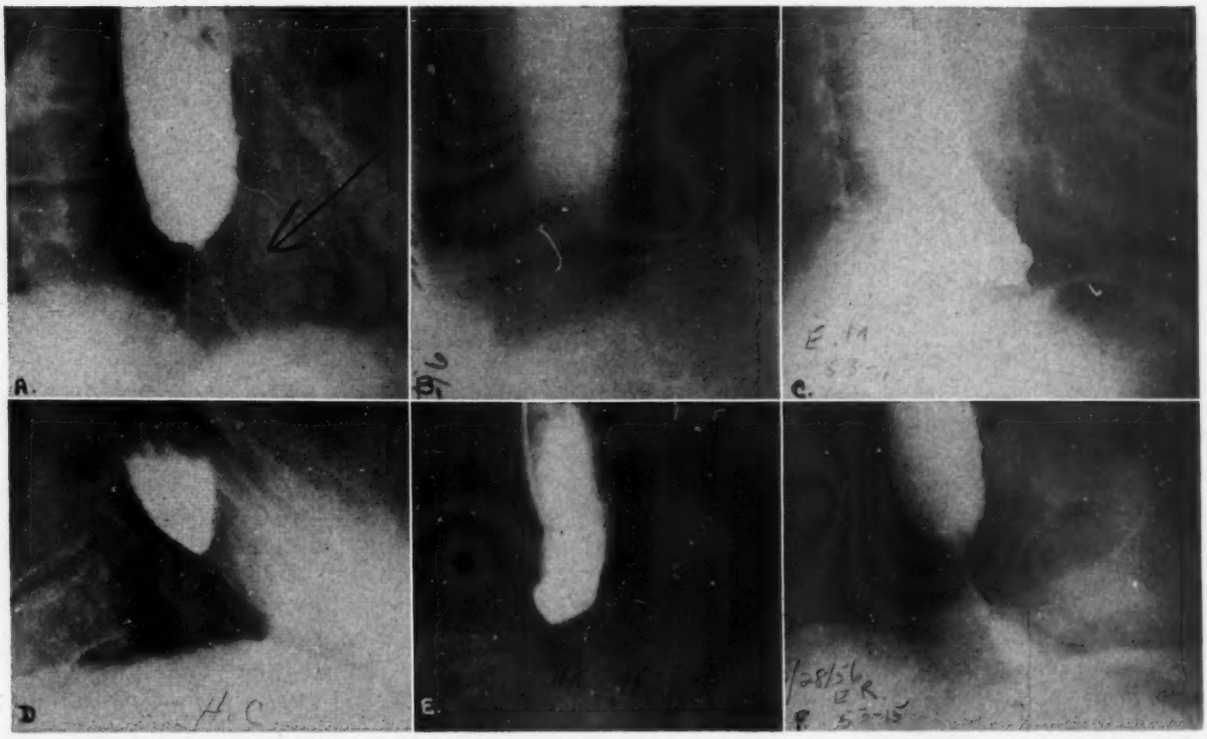

FIG. 5. Smooth conical narrowing, lower third of esophagus, with dilatation of esophagus above: (A) carcinoma of the esophagus, (B) recurrence of carcinoma at esophagojejunostomy, (C) achalasia, (D) carcinoma of esophagus, (E) achalasia, (F) carcinoma of fundus of stomach and lower end of esophagus.

they may be visualized by esophagoscopy. There are several reasons underlying this failure to demonstrate the ulcer roentgenologically, some of which may be shallowness of the ulcer and technical factors such as projection of the rays or poor or incomplete filling of the esophagus at that level.

Ulceration, *per se*, is rarely the only finding in malignant tumors, although superficial ulceration may occur in large tumors of the exophytic type. It is sometimes difficult to differentiate the superficial ulceration from the marked irregularity of the luminal surface of the tumor and vice versa.

Sinus or channel formation may be due to neoplastic or inflammatory extensions, congenital malformations or perforation caused by a foreign body.

Channel formation in the adult, which occurs as a result of perforation of a lesion, is usually due to carcinoma.

Congenital malformations of the esophagus in infants are frequently associated with esophagotracheal or bronchial fistulas. One form of congenital malformation is the rarely seen reduplication or double esophagus. The rudimentary portion may communicate with the main esophageal lumen as a pouch or channel formation, but more frequently is closed off at both ends and then acts as a mass or cyst in the mediastinum.

In perforations resulting from a foreign body or spontaneous rupture of the esophagus following severe vomiting, the history usually suggests the diagnosis and contraindicates the administration of barium, although occasionally lipiodol may be used to confirm the clinical impression or establish the site of perforation.

Pseudodiverticulum formation results from multiple adhesions to the wall of the esophagus in some forms of adhesive pathologic processes of the mediastinum. This fixation and interference with the contraction of the musculature may lead to formation of a pseudodiverticulum or multiple diverticula. Some authors designate this as "curling" of the esophagus.

In other conditions in which there is interference with the normal neuromuscular mechanism of the wall of the esophagus, irregular spastic segments alternate with formations of multiple pseudodiverticula which remain fairly

constant as to location and configuration for months or years. This has been designated as a "corkscrew" type of esophagus.

5. Margin of the Lesion. Irregularity or smoothness of the contour of the local lesion has already been discussed with the previous types of narrowing and filling defects. When the irregularity involves almost the entire contour of the esophagus, the lesion may be the result of chronic esophagitis or it may be secondary to extensive chronic mediastinitis. This type of lesion may also occasionally be found in extensive neoplasms with multiple fine ulcerations. Smoothness of the entire contour of the esophagus with lack of, or decreased, peristalsis is rare but may be seen in scleroderma.

6. Shortening of the Esophagus. This finding is usually associated with esophageal hiatus herniation of the stomach. Although various classifications of this condition exist (Harrington [*12*], Allison [*2*], Barrett [*3*] and others), that of Thomsen [*28*], which is a modification of Åkerlund's earlier classification, will be used here as a basis of discussion.

In the paraesophageal type of hiatus hernia only the stomach is involved in the herniation and the esophagus is essentially normal. The problem here is that of demonstrating the course of the esophagus. This may be difficult or impossible to do, as it enters the stomach below the level of the diaphragm.

Sliding hiatus hernias are of two types, those with and those without esophageal changes. In the former are included those cases in which the esophagus shows evidence of narrowing as a result of fibrosis and reflux esophagitis, peptic esophagitis or the formation of peptic ulcer [*8*]. The esophagus opens into the herniated portion of the stomach, either at its apex or its side, above the level of the diaphragm. In the second group, that is those without esophageal changes, the esophagus is only relatively shorter than normal and this finding may be quite insignificant. The sliding hernias are usually small although medium and large ones may occasionally be found. The differentiation roentgenographically between atypical phrenic ampulla and a small sliding hernia of the stomach may be extremely difficult, if not impossible [*23*].

The problem of congenital shortening of the esophagus has been pushed into the background by the reports of numerous workers which show that the usual case of relatively short esophagus and herniation of the stomach is probably not congenital but rather is the result of protracted reflux esophagitis with ensuing fibrosis and contraction of the esophagus. This type of hernia with a short esophagus is apparently the group in Thomsen's classification of sliding hernias in which the esophagus enters the apex of the herniated portion of the stomach, showing changes in the lower esophagus, as described previously. The true congenitally short esophagus presents primarily a condition for esophagoscopic diagnosis, since in such cases the esophagogastric junction cannot be determined on the x-ray film.

Occasionally, shortening is caused by an infiltrating scirrhous type of carcinoma in the region of the esophagogastric zone [*15*]. This may cause enough shortening of the gullet to result in herniation of the stomach through the esophageal hiatus in addition to other changes in the esophagus such as narrowing or dilatation above the lesion. The problems of the esophagogastric zone are further complicated by other conditions which are indicated in Chart III, M, N, and in Figure 6.

It should be noted that when a malignant type of tumor is suspected in the esophagogastric zone, its exact site of origin is not the most important factor to consider. When a lesion involving either the lower end of the esophagus or the cardiac portion of the fundus of the stomach is suspected of being malignant, the essential consideration is rather that it be very carefully studied to exclude or reveal involvement of the adjacent structure. Meticulous fluoroscopic study of the lower end of the esophagus in early malignant lesions may show deviation or splitting of the barium stream as it enters the cardia of the stomach. This is important in differentiating early malignant lesions from other types of lesions in this area. It is important to realize that microscopic infiltration of the tumor may occur across this esophagogastric zone without telltale evidence on the roentgenographic study.

7. Elongation of the Esophagus. This is usually a secondary effect and is frequently caused by a benign process. In addition to the marked dilatation in achalasia there is also redundancy and elongation of the esophagus in extreme cases.

8. Displacement of the Esophagus. Displacement as the result of an enlarged left atrium, tortuosity and elongation of the aorta, or

FIG. 6. Lesions with similar findings in lower end of the esophagus and esophagogastric zone: (A) carcinoma of the esophagus, (B) reflux esophagitis, (C) esophagitis and mild stenosis, (D) carcinoma of fundus of stomach and esophagus, (E) carcinoma of fundus of stomach and esophagus, (F) Schatzki ring(?) and small hiatus hernia.

anomalies associated with the great vessels and their abnormal course, produce fairly characteristic changes in the barium-filled esophagus which often lead to a specific diagnosis. Mediastinal masses such as carcinomatous lymph nodes, lymphosarcomas, inflammatory lymph nodes and masses [*13*], aberrant thyroid tumors [*10*] and other neoplasms or cysts help in the delineation and location of lesions which either are suspected clinically or have been observed on survey films. However, since the changes produced on the barium-filled esophagus are not specific, the final diagnosis is dependent on other roentgen and clinical findings, and laboratory and pathologic data. (Fig. 7.)

9. Mucosal or Relief Pattern. In extramucosal intramural lesions of the esophagus such as benign tumors the stretching of the mucosa over the lesion obliterates the folds and there is a narrowing or compression of the lumen of the esophagus at this site. Schatzki and Hawes [*22*] have clearly described the roentgen findings in extramucosal intramural lesions of the esophagus. In the profile view this lesion shows a very sharp angle where the edge of the tumor meets the uninvolved wall of the esophagus. When seen *en face* the lesion will be sharply outlined in the relief picture of the esophagus. The characteristic features may be obscured and not demonstrable when there is too much or too little barium present in the lumen of the esophagus. Although this organ appears markedly narrowed in the involved area, there is usually no obstruction to the passage of the barium; and as a rule the large intramural tumors narrow the esophagus only in one direction while they stretch and widen it in the other. Profile views of extrinsic or unattached tumors of the esophagus show a gentle slope from pressure, with no sharply angled corners. In frontal views the defect produced

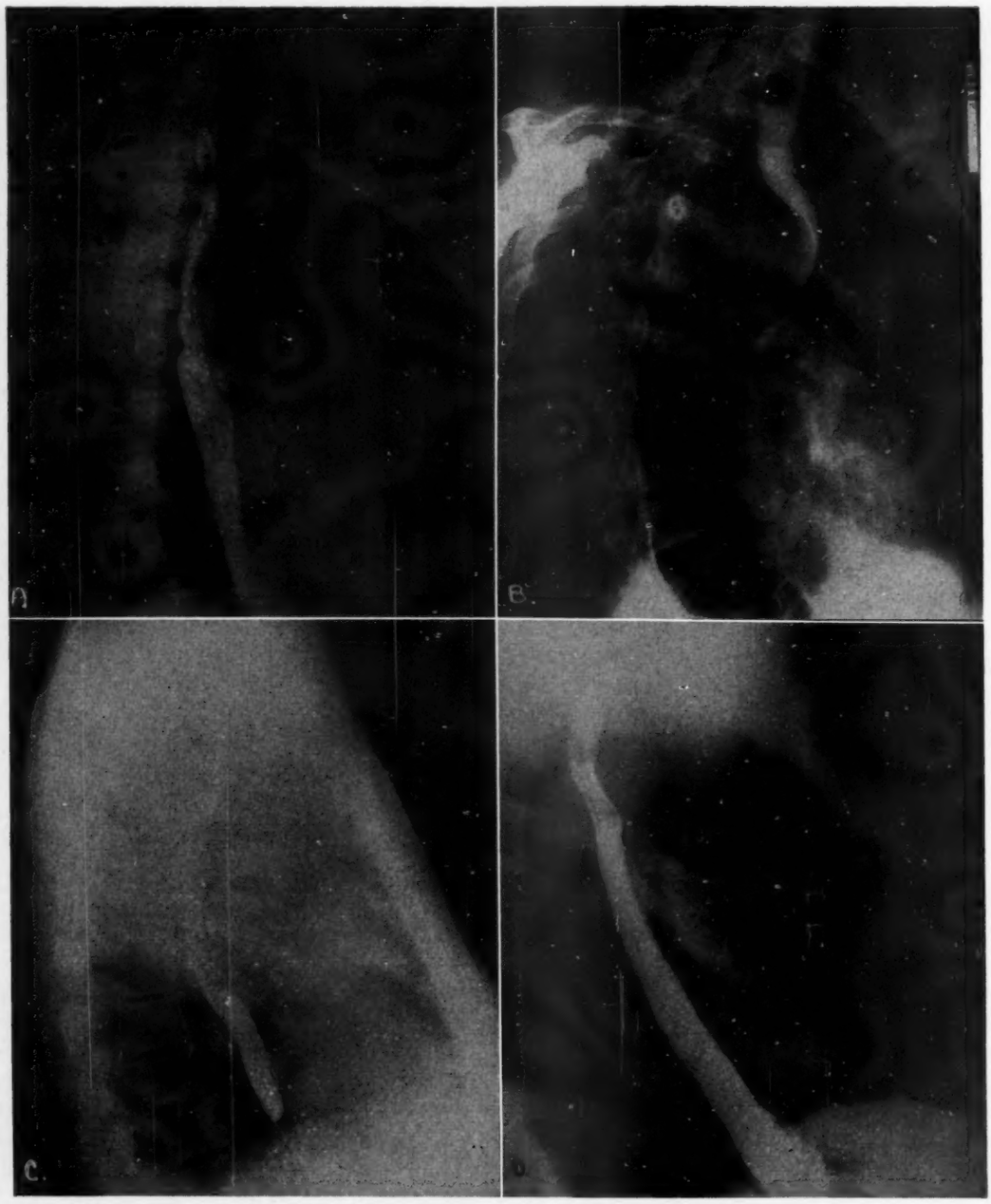

FIG. 7. Compression and displacement of the esophagus: (A) bronchogenic carcinoma with involvement of the mediastinum, (B) aneurysm of aorta, (C) lymphosarcoma of lungs and mediastinum, (D) aberrant thyroid.

by these extrinsic tumors is as a rule poorly defined, with blurred edges. It is to be noted that extraesophageal lesions which are firmly attached to the esophageal wall may simulate intramural lesions.

In exophytic malignant tumors of the esophagus the mucosal pattern is replaced by irregular intrusion of the malignant tissue, which not only erases the normal folds but also replaces them with an irregular surface which when coated lightly with contrast material gives the appearance of a disordered pattern. Other conditions such as severe edema, erosions, granulation tissue and excrescences may also produce similar disordered patterns of the mucosal relief surface. In varices the engorged submucosal veins vary in fullness according to the degree of existing intra-abdominal pressure, as well

as intraluminal pressures caused by different technics in filling the esophagus. These also produce a change in the relief pattern which sometimes simulates a row of beads in configuration or resembles worm-like structures or large, irregular, coarse folds. The demonstration of the varices radiologically is far from satisfactory, considerably less than 50 per cent of the proved cases being detected by roentgen method. In very early esophageal varices the roentgen demonstration is rarely possible. Very occasionally, esophageal varices are demonstrated by roentgen method in clinically unsuspected, asymptomatic cases.

10. Functional Motor Activity. As mentioned previously, this phase of roentgen diagnosis is fraught with many more uncertainties and is more prone to misinterpretation than is diagnosis of organic lesions. The evanescent nature of muscular activity and the variability and rapidity of progression of peristalsis, especially in the upper portion of the esophagus, make fluoroscopy the usual method of study of these phenomena. Disorders of motor functional activity are usually more significant at the pharyngoesophageal and esophagogastric zones. In the pharyngoesophageal region, functional disability in such conditions as Plummer-Vinson syndrome, pseudobulbar palsy and other neuromuscular disturbances are relatively more common in women than in men and usually denote a benign lesion in the esophagus. Occasionally, however, motor dysfunction may obscure an underlying or concurrent malignant lesion, inasmuch as the difficulties encountered in demonstrating the functional lesion satisfactorily at this site are also responsible for the poor delineation of an organic lesion on roentgen film. It is to be noted that cancer involving the lower two-thirds of the esophagus occurs more frequently in men than in women [29]; however, the reverse is true in carcinoma in the upper third of the esophagus or the pharyngoesophageal zone, and here apparently it is associated with Plummer-Vinson syndrome (sideropenic anemia). Disordered motor function of the esophagogastric zone is usually encountered in achalasia. The mechanism need not be discussed here, but it is worth while mentioning that although antispasmodics have no appreciable effect on this condition the administration of amyl nitrite may occasionally produce temporary relaxation which may be helpful in differential diagnosis. The mildly dilated esophagus with smooth conical narrowing, as previously mentioned, is occasionally found in carcinoma.

11. Associated Roentgen Signs in Adjacent Structures. As a rule, signs in adjacent structures are demonstrated on survey films of the chest and mediastinum without the aid of barium in the esophagus. Such findings as air in the mediastinum or hydropneumothorax, usually on the left side, are associated with perforation of the esophagus. In cases of suspected perforation it is better to use lipiodol or similar substances rather than barium, as mentioned previously.

12. Modification of Signs by Special Procedures. Some opaque foreign bodies are fairly obvious in their diagnosis; others require special technics to demonstrate their location or the site of the obstruction. Many foreign bodies become impacted in the upper portion of the esophagus adjacent to the pharyngoesophageal zone; and since this zone is in the region of the shoulders, it is an area in which it is most difficult to demonstrate the slightly radiopaque foreign bodies such as fine chicken bones or the non-opaque fish bones. Special technics which may be used consist of the administration of either thick barium paste to coat the foreign body or barium-filled capsules or impregnated cotton or bread to show the site of the narrowing or the obstruction. In some cases esophagoscopy may be necessary to exclude a suspected foreign body which cannot be demonstrated by x-rays.

SUMMARY AND CONCLUSIONS

The major intrinsic and extrinsic lesions affecting the esophagus have been presented in terms of their descriptive roentgen findings or signs. The differential diagnosis of these lesions has been discussed with the aid of diagrammatic sketches of the roentgen findings, corresponding charts and x-rays. It was noted that the same lesion may have various types of roentgen findings depending on the stage of the disease, the progression of the process or the sequelae produced.

In some cases the roentgen signs are so characteristic that they are considered pathognomonic of a particular lesion, whereas in many others lack of specificity or the similarity of roentgen findings makes it imperative to correlate the roentgen findings with all the available data, the clinical picture, endoscopic

study and often the biopsy report, before a definitive diagnosis can be made.

Certain problems in the study of esophageal lesions by roentgenology exist because of inherent drawbacks of the method. Other problems such as functional motor activity arise because of the tubulomuscular characteristics of the esophagus. The controversial pharyngoesophageal and esophagogastric zones, which are the site of many of the problems, have been briefly considered.

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Esophagoscopy in the Diagnosis and Treatment of Esophageal Diseases

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THE endoscopist plays a most important role in the diagnosis and treatment of diseases of the esophagus, and has become recognized as an indispensable member of a combined team which includes the gastroenterologist, roentgenologist and the surgeon.

Through the generalized use of endoscopic procedures, the interior of the esophagus has become one of the most readily examined areas of the body. Direct visualization of the esophagus permits the detection of incipient as well as advanced disease as it appears in living persons. In combination with the roentgenologist, the esophagoscopist has contributed much to clarify some important aspects of esophageal physiology including motor activity of the terminal portion of the esophagus. In addition, motor changes in the esophagus resulting in generalized medical diseases have been visualized and studied by esophagoscopy.

In actual clinical practice, the role of the endoscopist is an important one in the diagnosis of esophageal disease. No matter how classic the roentgen findings and clinical history may appear, final confirmation by direct visualization and biopsy is most desirable. In instances of neoplastic invasion, the exact site of the tumor and the condition of its proximal mucosa can best be ascertained by endoscopy.

It is well known that in esophagoscopy there is an inherent risk in the procedure depending on the skill and experience of the specialist. Except in instances of severe debility, marked cervical kyphosis or inability to open the mouth wide as in dermatomyositis, this minimal risk should not deter one from esophagoscopic examination.

PRACTICAL ANATOMY

The esophagus is a tubular muscular organ which connects the pharynx above with the stomach below. For practical purposes it is most convenient to divide the organ into three portions: the upper esophagus, mid-esophagus and terminal esophagus.

The upper esophagus begins at the lower limit of the pyriform fossa where it leads into the cricopharyngeal region. This is at the level of the sixth cervical vertebra. It is to be recalled that the musculature of the upper third of the esophagus is striated and under voluntary control, and the mouth of the esophagus relaxes only when a bolus of food passes from the hypopharynx into the esophagus or during vomiting or eructation. The inferior constrictor muscle which controls this area is inserted into the median pharyngeal raphe and arises from the posterior borders of the thyroid cartilage. The cricopharyngeal or oblique portion of the inferior constrictor muscle arises from the lower portion of the cricoid plate and is also inserted into the median raphe. There is a decussation between these two muscle bodies which constitutes a weak portion through which pulsion diverticula may originate.

This area is of extreme importance to the endoscopist and to the surgeon because it is the most frequent site of instrumental perforation, as well as the most common site for the arrest of esophageal foreign bodies.

The cervical esophagus is enveloped in the deep layer of the deep cervical fascia which is that same layer which surrounds the prelaryngeal muscles, larynx, thyroid gland and trachea. Behind the esophagus is the prevertebral fascia which is a loose layer of areolar tissue extending down into the mediastinum.

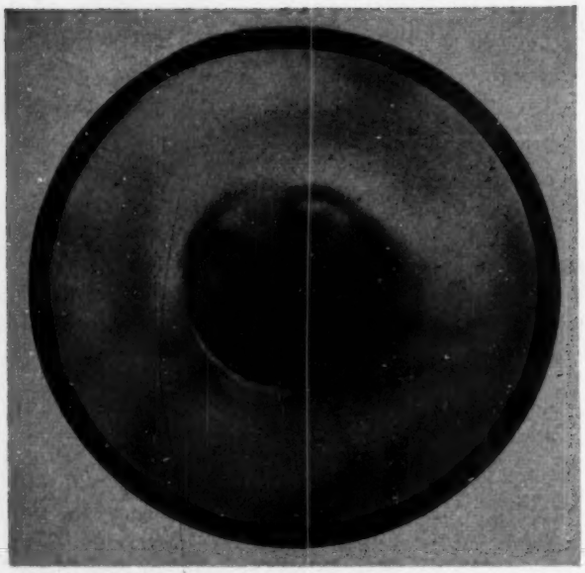

FIG. 1. Endoscopic appearance of the rosette. This is formed by contraction of the inferior esophageal sphincter and is usually found at 38 cm. from the upper incisor teeth.

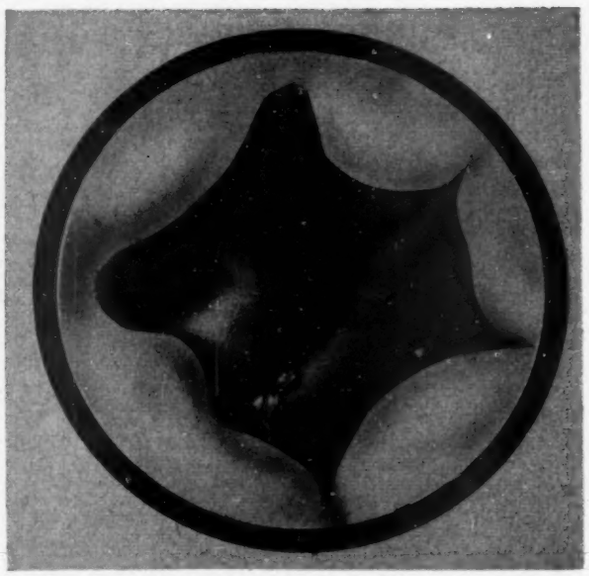

FIG. 2. Endoscopic view of vestibule at the Z-line or gastroesophageal juncture. The pale esophageal mucosa is distinct from the crimson rugae distally.

Along this loose pathway infections may rapidly descend into the thorax, making this tract of great practical importance in instrumental as well as foreign body perforations.

The mid-esophagus is freely movable. In general direction it extends downward anteriorly and to the left toward the diaphragmatic hiatus. Although the upper esophagus is located in the midline, in the lower neck it passes over slightly to the left and then at the level of the fifth dorsal vertebra it returns to the midline. Below this it passes to the left and anteriorly until it reaches the diaphragm.

In its descent the thoracic esophagus is in relationship to the great vessels, pericardium and left main bronchus. These may produce indentations in the esophagus which may occasionally be seen roentgenologically and esophagoscopically. The most constant of these is at the level of the fourth thoracic vertebra where the arch of the aorta causes a compression of the left side of the esophagus as it passes posteriorly.

Immediately below this, the left bronchus may produce an anterior compression of the esophagus but this is rarely seen through the esophagoscope. The esophagus below this and down to the level of the diaphragm is in contact with the pericardium, but no indentation is produced except by the left auricle which produces so shallow an indentation that it is indistinguishable through the esophagoscope.

The major constrictions of the esophagus are: (1) at the region of the cricopharynx, (2) at the arch of the aorta and (3) in that region just above the diaphragm which is termed the inferior esophageal sphincter and which is recognized endoscopically as a rosette. These are 16, 23 and 38 cm., respectively, from the upper incisor teeth.

The muscle walls of the esophagus are an outer longitudinal and an inner circular. As stated previously, these are striated voluntary muscles in the upper esophagus and become involuntary or smooth in the mid- and lower esophagus.

It is important to remember that the esophagus has no serosa in contrast to the stomach which has a peritoneal covering. The adventitia or the outermost layer of the esophagus is merely a fibrous layer condensed from the mediastinal connective tissues. Because of its consistency as loose areolar tissue, it offers little resistance to perforation. In addition, suturing of the outer layer of the esophageal wall is more difficult because of the absence of a serous layer when the thorax is open. The esophagus may be differentiated from the cardia of the stomach, which may be in the thorax, by the fact that it is uncovered by peritoneum.

The motor activity of the terminal esophagus

has recently been the subject of great interest and considerable amounts of research and speculation. The last word in the clarification of its anatomy and physiology has by no means been uttered. This is particularly true since the findings of the anatomist, esophagoscopist and roentgenologist and surgeon have not been completely correlated.

ESOPHAGOSCOPIC APPEARANCE

The normal esophageal mucosa is smooth, glistening and has a yellowish pink color. The intensity of the color is dependent upon the character of the illumination. Nevertheless, both with distal and proximal lighting, the esophageal mucosa has a distinctive appearance and can readily be distinguished from the crimson rugae of the cardia. Normally there is an expansion and contraction of the thoracic esophagus with inspiration and expiration. As the region of the gastroesophageal junction is approached, one finds a contraction of the esophagus which makes it apparent that the passage between the resting esophagus and the stomach is normally not patent. In addition to the protection offered by the angular insertion of the tubular esophagus into the stomach distally, a muscular closure of the terminal segment of the esophagus aids in preventing reflux of gastric juice. This sphincteric action is recognized in esophagoscopy as a distance of 38 to 39 cm. from the upper incisor teeth as a rosette formation in the mucosa terminating in a dimple. (Fig. 1.) This rosette corresponds to the inferior esophageal sphincter.

Except under conditions of vomiting, this closing mechanism is in tonic contraction and represents a bar between the negative pressure in the esophagus and the positive pressure in the stomach. Until the tube is introduced beyond this mechanism, gastric juice is not obtained.

Gentle pressure on the rosette in the direction of the dimple will cause the folds to relax and the esophagoscope glides into the vestibule or sphincter canal. The proximal 1 to 1.5 cm. of this canal is lined by esophageal mucosa and just distal to this, the Z-line or actual junction of gastric and esophageal mucosa is recognized. (Fig. 2.) This junction can be determined more readily by esophagoscopy than by roentgenology. The entrance of the tube beyond the Z-line is heralded by the escape of gastric juice and the appearance of the crimson gastric rugae. A few millimeters beyond this the widened pouch of the stomach proper is identified.

FIG. 3. Barium swallow in a patient with a small sliding hiatal hernia showing delay in the passage of the barium column by a short localized constriction which corresponds to the rosette.

The rosette corresponds to the level of the temporary arrest of barium often seen above the level of the diaphragm when the patient is in the reclining position and swallowing. (Fig. 3.)

No description of the terminal esophagus is complete without a discussion of its interrelationship with the diaphragm. First, it must be recalled that a small segment of the terminal esophagus lies below the diaphragm. This segment enters the stomach at an acute angle and is one of the factors that prevents regurgitation of acid gastric juice into the esophagus. Second, the supporting and constricting action of the sling formed by the decussation of the tendinous fibers of the right crus of the diaphragm also helps to prevent regurgitation, particularly on deep inspiration.

DIVERTICULOSIS

Pulsion diverticula occur most often posterior to the cricopharyngeal area. The diverticulum is caused by the mucosa protruding through the muscle-missing space of Lannier-Hackerman in the posterior line between the hypopharynx and the esophagus.

The herniation forms a pouch alongside the esophagus which displaces the lumen of the esophagus proper anteriorly. It also kinks the esophagus so that it is very difficult to locate the esophageal lumen through the esophagoscope and to enter it with the tube. On the other hand, the diverticular pouch is readily entered with the esophagoscope.

As the diverticular sac increases in size, it produces pressure on the esophagus and dysphagia becomes troublesome. As a result of overflow from the diverticulum, aspiration into the tracheobronchial tree with its sequelae is a common occurrence. It is often difficult to locate the sac surgically and esophagoscopy is used as an aid to the surgeon in defining the sac by illuminating its interior.

Mid-esophageal diverticula or traction diverticula usually arise adjacent to the pulmonary hili and spring from the lateral wall of the esophagus. The majority extend to the left and anteriorly. They rarely grow posteriorly. These lesions seldom retain food or barium, and therefore, do not often produce symptoms. They are said to be due to the pull of adherent tuberculous mediastinal lymph nodes.

Epiphrenic diverticula develop from the ampulla or area just proximal to the hiatus and are considered congenital in origin.

The diverticulum takes the form of a spherical pouch with moderate narrowing at the neck. The majority extend to the right and anteriorly. These lesions have serious complications which are chiefly bronchopulmonary and due to massive or repeated small aspirations.

Esophagoscopically, a shelf is seen just proximal to the esophagogastric junction. This shelf leads into a blind pouch on the side, whereas the esophageal lumen continues on in a straight direction.

CARDIOSPASM OR ACHALASIA

Esophagoscopy should always be performed at least once in every suspected case of cardiospasm. No matter how characteristic the clinical history and roentgen findings may seem to be, diagnostic endoscopy is indicated for confirmation because differentiation for peptic cicatricial stenosis or gastric carcinoma is often impossible without direct visualization and biopsy.

In cardiospasm, the findings on endoscopy vary from mild widening to marked dilatation with retention and stagnation of food. After removal of the retained secretion, marked dilatation and tortuosity of the esophagus with inflamed mucosa is seen. In the distal esophagus, areas of non-specific esophagitis and leukoplakia may be noted. In some cases, elongation of the esophagus as well as dilatation takes place. This is important to recognize because in a markedly dilated and elongated esophagus the rosette may be extremely difficult to locate. However, once it is found, gentle pressure in the dimple will cause the inferior esophageal sphincter to relax and the vestibule is entered. If the stomach is not entered readily, the existence of neoplastic mucosa should be suspected and a biopsy specimen taken.

Endoscopic treatment of cardiospasm consists of either passive dilatation with Hurst mercury-weighted tubes or active dilatation with the pneumatic or hydrostatic dilating bags. The results of the former are transitory and disappointing and since the dilatation must be performed before each meal, it is very distasteful to the patient.

By means of the pneumatic bag, a real stretching of the esophagus is accomplished. The cardioesophageal junction is forcibly dilated to 4½ to 5 cm. If pain is not produced, the treatment is usually ineffective. This accomplishes a separation of the muscle fibers which may rarely result in an extravasation of air into the mediastinum or under the diaphragm. Although danger exists in this method of therapy, serious accidents are rare and should not preclude the use of this form of treatment.

It is to be remembered that the results of any form of treatment of cardiospasm are disappointing and seldom end in cure. Several active dilatations may be necessary because of the return of symptoms.

FOREIGN BODIES

Foreign bodies in the esophagus are a common occurrence. Whereas foreign bodies in the

stomach or intestines may be expected to pass through the alimentary canal, this is not true of the esophagus. When a foreign body becomes impacted in the esophagus, edema and reaction tend to hold it more firmly and also to make esophagoscopic examination and manipulation more difficult. Therefore, it becomes an urgent matter to remove these foreign bodies as soon as their presence is diagnosed. If they cannot be detected preoperatively by x-rays but are strongly suspected, esophagoscopy should be done as a diagnostic procedure.

The greatest percentage of foreign bodies are arrested in the upper esophagus just below the cricopharyngeal area. This occurs because the strong pharyngeal muscles propel it this far and the weaker esophageal muscles cannot propel it further. There are also heavy redundant folds in this region which may entrap a foreign body.

It is important to remember that foreign bodies are frequently arrested by strictures or webs of the esophagus, and diagnostic esophagoscopy and radiology are called for in any case of recurrent foreign body impaction in the esophagus.

The complications of foreign bodies in the esophagus are chiefly perforation and infection. A bone or other object lodged in the esophagus for longer than a short period of time may cause pressure, necrosis, perforation and fulminating infection of the retropharyngeal space with or without descent into the mediastinum. Foreign bodies have been known to migrate through the esophageal wall and end up in the thyroid, lung or mediastinum. They have also been known to perforate the great vessels of the thorax and the bronchi.

Fortunately, antibiotics have proved an invaluable aid in the treatment of these complications. However, the mechanical removal of these foreign bodies must still be carried out. The endoscopic technic of the removal may be found in any of the standard monographs on esophagoscopy. External removal may, of course, occasionally have to be performed if perforation has already resulted.

It is convenient to mention here that spontaneous rupture of the esophagus occurs, although rarely. It may follow severe retching and vomiting and occurs in the vast majority of patients in the lower esophagus. If the diagnosis is not made readily and prompt surgical and antibiotic therapy instituted, death follows regularly.

INFLAMMATORY DISEASE OF THE ESOPHAGUS

Non-specific inflammatory disease of the esophagus occurs in the region of the cricopharyngeus muscle which constitutes the proximal sphincter. The more common lesions in this location are foreign bodies, traumatic perforation, chronic hypopharyngitis and decubitus ulcer.

Instrumental perforation of the esophagus is most frequent in the hypopharynx. In most instances, the esophagoscopist is immediately aware of having produced a tear or false passage. Brisk bleeding and the loss of normal landmarks should arouse the suspicion of perforation. The administration of large doses of antibiotics, sedation and parenteral feedings will usually prevent a descending mediastinitis. After a short interval, the classic syndrome of fever, dysphagia, crepitation, severe interscapular pain and x-ray evidence of subcutaneous emphysema establishes the diagnosis. Experience has proved that conservative therapy, as outlined, will prevent further complications in the great majority of cases. Occasionally cervical drainage of a localized periesophageal abscess will be required. Esophagoscopic manipulation is contraindicated.

On rare occasions, the signs of perforation and a periesophageal infection may not become manifest until several days after endoscopy. This is especially prone to occur in elderly patients with cervicodorsal kyphosis or when the patient is unable to open the mouth widely because of fixed bridgework. Prolonged pressure of the metal tube against the esophageal mucosa overlying a prominent cervical spine or spur may be sufficient to cause a pinpoint area of necrosis and subsequent perforation. Such pressure may inadvertently result from an attempt to expose the cardia by depressing the head and directing the esophagoscope anteriorly. Because of the slight inherent risk of such complications, all patients undergoing esophagoscopy are given antibiotics for one or two days after the examination.

Chronic hypopharyngitis is often associated with hypochromic anemia, glossitis, achlorhydria and iron deficiency. This so-called Plummer-Vinson syndrome occurs predominately in females and is frequently accompanied by dysphagia. The difficulty in swallowing may

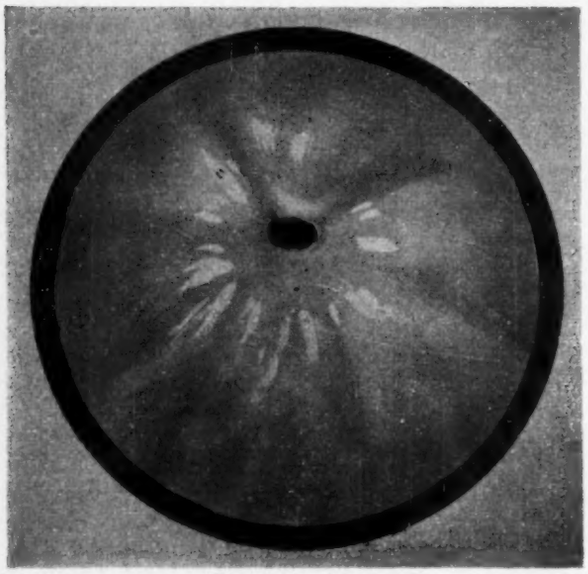

FIG. 4. Endoscopic appearance of cicatricial stenosis with eccentric location of narrowed lumen.

result from the formation of a narrow stricture or web at the opening of the esophagus. The web is formed by a thin layer of chronically inflamed mucosa and can easily be dilated by the passage of a bougie or an esophagoscope. Such dilatation not only gives prompt relief of the dysphagia but also affords marked improvement in the general condition of the patient.

Decubitus ulcer of the postcricoid region may result from the prolonged use of a Levin feeding tube. Such tubes have frequently been employed in patients undergoing extensive gastrointestinal surgery who require prolonged bedrest. With the patient in the reclining position, the pressure of the overlying cricoid cartilage against the rubber tube may cause ulceration of the hypopharyngeal or esophageal mucosa. In one specific instance of a four year old child, a Levin tube had been allowed to remain *in situ* for four weeks. An ulceration of the posterior surface of the cricoid cartilage resulted in a slough into the larynx. In most instances, the symptoms of dysphagia and pain occur four to six weeks after withdrawal of the tube. By this time a stricture has developed. At endoscopy, a pinpoint lumen may be encountered surrounded by a fibrotic stricture which requires repeated dilatations.

STRICTURE

Congenital stricture of the esophagus may be single or multiple and may involve a larger or smaller segment of the lower esophagus. Often such incomplete or partial stenosis may not become manifest until solids are added to the child's diet. A bolus of unmasticated food may become embedded in the narrowed lumen and produce complete esophageal obstruction. A non-specific esophagitis may accompany such stenosis. Gradual dilatation with graded bougies is the treatment of choice. Usually a rather wide segment of the lower esophagus is involved in congenital stricture.

CICATRICIAL STENOSIS

The accidental swallowing of caustics is the most frequent cause of cicatricial stenosis. Less common causes are specific ulcer of the esophagus, embedded foreign bodies and healed peptic ulcerations. The cicatricial stenosis is the result of submucosal fibrosis of the deficient mucosal surface. The deeper the ulcer, the more pronounced the resulting fibrosis. In such instances, the epithelial surface may regenerate and the only suggestion of the previous ulceration is the clinical history. Endoscopically, the cicatricial area has a paler color than the normal mucosa. The narrowed lumen is most often eccentrically placed and that part of the circumference of the mucosa involved in the cicatrix seems whitish. (Fig. 4.) If the stenosed segment is short, it may be possible to look beyond into the distal esophagus. When the mucosal surface is intact, one must consider caustic stricture, healed peptic stenosis, scleroderma or external compressive stenosis as diagnostic possibilities.

TREATMENT

The immediate treatment of caustic burns consists of neutralization of the swallowed chemical and supportive measures. After the initial acute phase has been overcome, it may be possible to prevent cicatrization by early passage of the Hurst mercury-weighted tube in graduated sizes. The swallowing of the soft rubber tube can be started on the third or fourth day after the chemical burn. Progressively larger tubes can be employed daily as tolerated. On or about the tenth day, a thorough inspection of the entire esophagus with an evaluation of the damage sustained should be made by peroral endoscopy. A search for areas of ulceration and narrowing will yield necessary information as to the sites of future strictures which may require treatment. The

Hurst tube may aid in preventing stricture formation but is inadequate for dilation of a fibrotic stricture. After ascertaining the location and extent of the stenotic strictures, dilatation therapy should be instituted promptly.

Dilatation of an esophageal stricture is best performed either by direct visualization through an esophagoscope or by guided bouginage over a previously swallowed thread. Blind bouginage is seldom indicated and should not be resorted to except with soft rubber mercury-weighted tubes.

The degree of success of dilatation which is possible is dependent largely upon the extent of the stenotic segment involved and the character of the fibrosis. In ascending fibrosis of the esophagus and in congenital stenosis, a rather long segment of the lower esophagus is fibrotic and dilatation becomes an arduous procedure with less promise of success. Conversely, in early lye stricture and peptic ulceration, an adequate lumen can be readily obtained in most cases by a few dilatations. The simplest type of stricture to treat is that which involves only the mucosa and submucosa of the esophagus and stretches across the lumen of the esophagus like a membrane. This applies particularly to the so-called web of the mouth of the esophagus associated with glossitis and secondary anemia. The mere introduction of the bougie beyond the web often produces a cure.

PEPTIC ESOPHAGITIS AND PEPTIC ULCERATION OF THE ESOPHAGUS

It is generally agreed that esophagitis and esophageal ulcerations are related to the irritating and digestive action of gastric juice on the susceptible esophageal epithelium. Gastric contents may enter the esophagus during the use of an indwelling feeding or suction tube, as a result of vomiting or during comatose states. Gastric juice may also regurgitate into the esophagus in the presence of hiatal herniation and in association with a peptic ulcer of the stomach or duodenum. (Fig. 5.)

While the color of the esophageal mucosa in any single patient is partially determined by the character of the illumination and other individual factors, the hue is uniform throughout the entire length. However, in peptic esophagitis, the change in color of the lower segment of esophagus is striking and a valuable diagnostic aid. The transition from the yellow-pink color of the proximal esophagus to the red, hyperemic color of the distal portion is characteristic of the esophagitis.

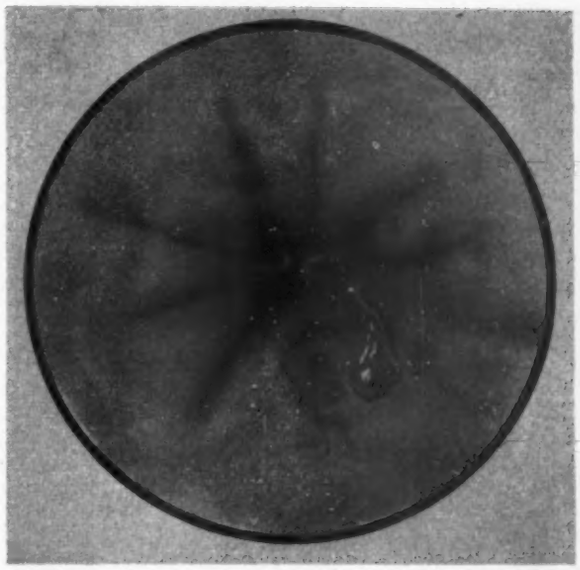

FIG. 5. Endoscopic view at gastroesophageal juncture in hiatal hernia with mild reflux.

Peptic esophagitis may occur with the sliding type of hiatal hernia and in association with duodenal or gastric ulcer. It may result from persistent vomiting or the prolonged use of an indwelling feeding tube. In such instances an ulcerative inflammatory process usually involves a long segment of the distal esophageal mucosa. Multiple erosions and areas of intense hyperemia interspersed with minute irregular ulcerations may extend along the distal 10 cm. of the esophagus. (Fig. 6.) Throughout this area of the esophagitis, a uniform narrowing of the esophageal lumen is encountered terminating at the gastroesophageal junction. There may or may not be regurgitation at the time of esophagoscopy and the rosette is apt to be 2 to 3 cm. above the diaphragm. In these cases the peptic esophagitis always occurs in the esophageal mucosa proximal to the gastroesophageal junction. Should spontaneous healing occur, a smooth fibrotic stenosis just above the gastroesophageal juncture may result.

SHORT ESOPHAGUS WITH MARGINAL ULCERATION

There is another type of peptic ulceration occurring in association with a hiatal hernia which is of the traction type and which does not have the appearance of a typical sliding hernia. In these cases heterotopic gastric

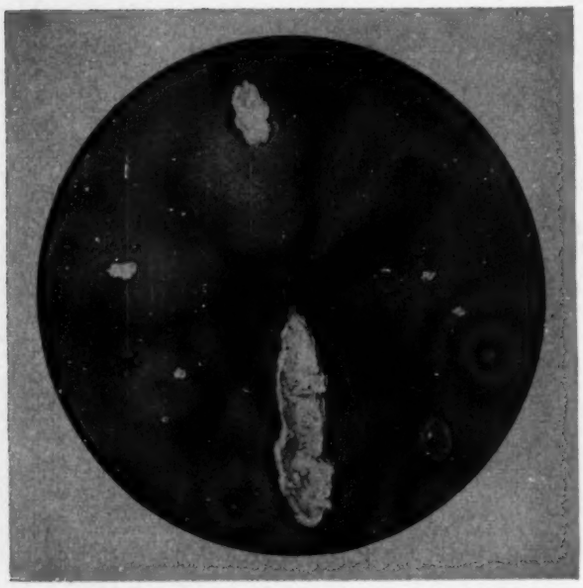

FIG. 6. Endoscopic view of peptic esophagitis showing superficial erosions, intense hyperemia and occasional hemorrhage.

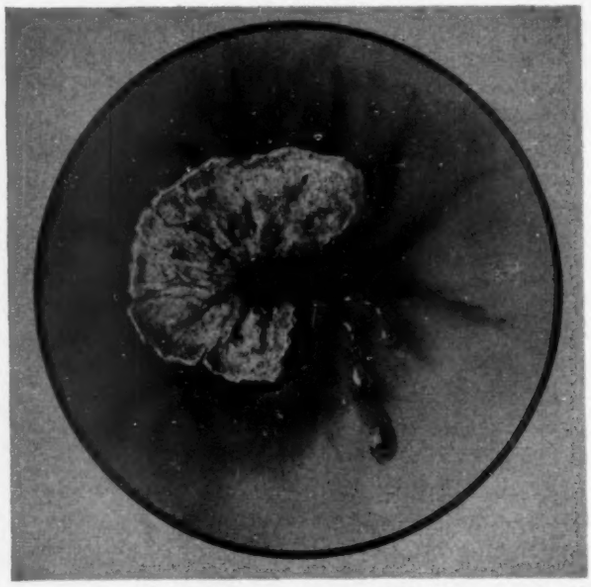

FIG. 7. Endoscopic view of marginal ulceration with constant reflux of gastric juice. Superficial ulceration can be seen in extreme distal portion of esophageal mucosa.

epithelium may or may not exist in the terminal segment of the esophagus. These cases of short esophagus with marginal ulceration are more apt to produce fibrotic stenosis.

Whereas normally gastric juice is not obtained until the gastric-lined portion of the cardiac canal is entered, gastric regurgitation is a constant feature of short esophagus with marginal ulceration. Reflux is encountered as soon as the tube enters the upper esophagus and continuous aspiration for proper visualization and the further passage of the tube is required. In short esophagus with marginal ulceration, the gastric juice can be seen to well up from below through the patent gastroesophageal juncture at the distal ulcer margin. The characteristic feature of the lumen at this point is its constant patency.

In the absence of gastric reflux, one rarely encounters significant endoscopic evidence of peptic esophagitis. Certainly, most patients with short esophagus and uncomplicated hiatal hernia show little evidence of esophagitis. When the gastroesophageal junction is competent, the esophageal mucosa appears grossly normal. Should incompetence develop at a later date and symptoms result, then the classic picture of peptic esophagitis is to be expected. In our experience, the patients who underwent endoscopy merely because of the roentgen finding of hiatal hernia had little evidence of esophagitis unless heartburn or other symptoms of regurgitation were present.

After thorough aspiration of the regurgitated esophageal contents, one can recognize the inflammatory changes in the lower esophagus with the erosion or ulceration distally.

The ulcerated area is covered by yellow-gray membrane or slough which can be readily removed by grasping forceps. (Fig. 7.) Occasionally, the attachment of this fibrinous cast is so tenuous that mere suction is sufficient to remove it with very little bleeding. A reddened, granular base is now visible. This is rather uneven, due to the presence of exuberant granulation tissue. A true ulcer crater with undermined edges and a necrotic base is rarely encountered. Rather, the erosion is superficial and the proximal edges less distinct than the distal which always borders on the gastric mucosa. Heterotopic gastric mucosa may be present laterally.

The lesion is frequently elongated and somewhat irregular with a tendency for encirclement of the distal 1 or 2 cm. of the esophagus. The yellow-gray membrane is not always present at both the initial and subsequent endoscopies. In such cases, the bed of the superficial erosion can be detected as an uneven, granular, reddish surface lacking the smooth epithelial covering of the normal esophagus. Minute mounds or elevations can be seen resulting from varying

loss of mucosal covering interspersed throughout the granulating surface.

Mucosal changes proximal to the ulcer are common and constitute the varying degrees of peptic esophagitis. Localized inflammation, congestion and edema with spotty, superficial erosions and hemorrhage occur. A narrow zone of hyperemia consistently surrounds the borders of the marginal ulcer and extends proximally up the esophagus for a variable distance. Although the appearance is quite characteristic, a routine biopsy must be done to exclude tumor.

The ulcerative phase is invariably accompanied by segmental spasm of the esophagus so that a visible narrowing of the esophageal lumen is encountered at the upper level of the esophagitis. This spasm, however, is usually overcome by the mere passage of the esophagoscopic tube. At the proximal margin of the ulceration, greater difficulty in the further introduction of the tube may be experienced due either to the presence of increased spasm or periesophagitis with fibrosis. The point of maximum narrowing is always at the gastroesophageal juncture in these instances. When fibrotic stenosis does occur, except in the rare case, the esophagoscope can push the narrowed gastroesophageal juncture for a distance of 3 cm. distally, thereby elongating the shortened esophagus. Gradual dilatation with graded bougies may be necessary before the tube can be finally introduced into the gastric pouch. Repeated esophagoscopic examinations in the same patient have demonstrated varying stages of the healing of the ulcer with occasional recrudesence. The gastric reflux which is the cause of the ulceration should be increased by the endoscopic dilatation; yet, paradoxically enough, the ulcer seems to improve by relief of the stenosis. If the lumen can be adequately maintained (non-obstructive phase) and the acidity controlled by antacids and anticholinergic drugs, there is a tendency for spontaneous healing. However, in marginal ulceration with hiatal hernia, healing is apt to be accompanied by fibrosis and contracture which may lead to varying degrees of stenosis and stricture formation. In one recent case, complete obliteration of the esophageal lumen resulted. In well advanced stenosis, the lumen may be constricted to 2 to 3 mm. as measured by the caliber of the bougie it can accommodate.

The diagnosis of peptic esophagitis and ulcer must be confirmed by endoscopic visualization and biopsy. Esophageal shortening as a result of spasm and fibrosis accompanying the ulcer occasionally occurs. Obstruction with marginal ulcer results either from segmental spasm during the acute phase or from cicatricial stenosis after healing.

Most cases of peptic stenosis of the distal esophagus are associated with short esophagus and hiatal hernia and result from previous ulceration. Narrowing of the esophagus is a relative term and must be evaluated by comparison with the average lumen of the esophagus. In the normal adult, an esophagoscopic tube with an outside diameter of 12 mm. is employed and is readily accommodated within the lumen, although the 16 mm. tubes may be tolerated. When the 12 mm. tube encounters a narrowing or stenosis, it simply implies that the diameter of the stricture is narrower than that of the tube. A relative stenosis can be said to be present when a 12 mm. tube cannot be passed except after preliminary dilatation with a Jackson bougie. Under such circumstances, a constriction as compared to the normal diameter of the esophagus certainly exists. Such findings on endoscopy can explain the occasional narrowing of the esophageal lumen, demonstrable by roentgen film in a patient who experiences no difficulty in swallowing a routine daily diet. Occasionally, a large bolus of food becomes embedded into the narrowed segment and may require esophagoscopy for removal. On the other hand, the endoscopist may encounter a band in a patient whose x-ray seemed within normal limits because the barium passed promptly into the stomach without any demonstrable delay. Direct visualization is the most important means of confirming the diagnosis.

PEPTIC ULCER IN AN ESOPHAGUS LINED BY GASTRIC MUCOSA

Whereas superficial erosions and ulceration of the esophagus are not uncommon, true isolated peptic ulcer is rare. Peptic ulcer of the esophagus occasionally occurs in instances of gastric mucosa–lined esophagus. The flat, heterotopic epithelium which surrounds the ulcer may not be distinguishable from the squamous epithelium except by biopsy. In one recent case, a penetrating ulcer of the esophagus was encountered in an elderly person. The ulcer was surrounded by a wide segment of velvety heterotopic gastric mucosa on the

esophageal side and gastric rugae distally. The proximal margin of the ulcer was very stenotic and could be dilated only with extreme difficulty. Multiple biopsies of the esophagus above the stenosis revealed heterotopic gastric mucosa. This is an instance of a Barrett type of ulcer.

In another indolent, peptic ulcer of the lower esophagus, a marked stenosis at the proximal margin made dilatation very difficult and hence visualization of the ulcer impossible. The ulcer was surrounded by squamous epithelium except at the lateral margins where gastric mucosa was in contact with it. The true peptic ulcer is more apt to be associated with stenosis at the proximal end of the lesion. This organic narrowing usually prevents proper visualization of the ulcer bed because of inability to insert the esophagoscope beyond the stenosis. A biopsy specimen taken deeply within the lumen of the stenosis may reveal the presence of necrosis, characteristic of the ulcer bed. The specimen of the surrounding mucosa usually shows an altered or heterotopic gastric mucosa. The endoscopic appearance in these cases may be that of a smooth cone-like narrowing with or without a superficial erosion proximally. The ulcer may occur at the juncture of the heterotopic gastric mucosa in the lower esophagus with gastric rugae, or may occur proximally at the juncture of squamous epithelium with the heterotopic mucosa.

The narrowing proximal to the ulceration is due to submucosal fibrosis which encircles the lumen of the esophagus and produces stenosis. In marginal ulceration or peptic esophagitis, the stenosis produced is more apt to involve a shorter segment of the terminal esophagus. With deeper ulcers, especially of the Barrett type, the fibrotic stenosis may involve a much longer segment of the lower esophagus. On esophagoscopy, a healed peptic stenosis has a smooth conical appearance tapering distally. The lumen may be as minute as 1 to 2 mm. on the first examination; usually dilatation can be accomplished with Jackson bougies through the esophagoscope up to No. 28 French. Further dilatation over a previously swallowed string can then be continued until a No. 40 French lumen is obtained. With ascending fibrosis of the esophagus, more likely associated with deep ulcer, the segment of esophagus involved in the stenosis is larger and bouginage is less effective.

STENOSIS FOLLOWING GASTROESOPHAGEAL SURGERY

Cicatricial stenosis following gastroesophageal surgery is occasionally encountered at the suture line. Since reflux of gastric juice is unavoidable after by-passing or resection of the cardia, peptic erosion may result when acidity persists. Indeed, after procedures such as esophagogastrostomy or ablation of the cardia for achalasia, peptic erosion has occurred so frequently as to make these procedures unpopular.

Marginal ulceration is said to follow limited resection of the stomach and esophagus for carcinoma of the cardia. This is a rare complication of esophagogastrectomy for carcinoma in our experience at Mt. Sinai Hospital. More commonly, stenosis at the suture line may be associated with what may be referred to as a "silk granuloma." The esophageal mucosa at the site of the silk suture becomes edematous and protrudes into the lumen of the gastroesophageal juncture. On endoscopy, the black silk suture can be seen between the folds of the inflamed redundant mucosa. After cutting the silk with a punch biopsy forceps and extraction of the suture, the condition promptly improves. Nevertheless, the edematous mucosa is removed with forceps and examined for evidence of recurrence of neoplasm.

Two instances of peptic stenosis following the Heller operation for cardiospasm have been encountered. Gastric reflux and marginal ulceration resulted after incision of the esophageal and gastric musculature down to the mucosa. Incompetence of the esophageal sphincter with regurgitation is much more apt to occur after the Heller operation in the presence of hiatal hernia with achalasia.

VARICES OF THE ESOPHAGUS

Varicose veins of the esophagus, associated with portal hypertension or obstruction of the splenic veins, can be more easily diagnosed by endoscopy than by radiography. The dilated varices distend the mucosa and can be detected easily under direct vision. They are much more common in the lower third of the esophagus but may extend throughout the entire length of the organ. Routine esophagoscopy with the rigid esophagoscope for detection of symptom-free varices is not recommended. Varicose distention and distortion of the gastric veins may also occasionally be demonstrated. It is important

to stress that erosion of the overlying mucosa and other evidences of recent hemorrhage such as adherent blood clots may be ascertained only by endoscopy. This, of course, cannot be shown roentgenologically. As a matter of fact, the x-ray can be relied upon to diagnose less than 40 per cent of the advanced cases of esophageal varices and early cases may be missed entirely.

In the proximal half of the esophagus the varices often appear as isolated bluish spheres surrounded by congested mucosa. In the distal third and more particularly around the gastroesophageal juncture, they may occur as clusters of soft bluish red tortuosities protruding into the lumen. The varicosities are easily compressible and offer no resistance to the further passage of the esophagoscope. Erosion of the superficial mucosa with adherence of old blood clots usually signifies the site of recent hemorrhage. The easy collapsibility of the varices may explain the difficulty in their demonstration by contrast media.

Esophagoscopy should be employed to corroborate the clinical or roentgen diagnosis of esophageal varices in patients with a history of recurrent hematemesis. The risk of initiating profuse bleeding from the varices is negligible provided, of course, that care is exercised. When varices are found in the esophagus, a search should be made for gastric varicosities since the type of therapy may be modified by the knowledge of their existence.

The treatment of esophageal varices by the injection of sclerosing solution has met with very limited success. The thrombosis of this important esophageal collateral circulation increases the portal hypertension and may favor the formation of gastric varices. When gastric varices coexist with esophageal varicosities, the injection treatment is contraindicated. Occasionally, however, the injection treatment has proved valuable following unsuccessful shunt procedures and splenectomy. Also, a patient whose general physical condition does not permit major surgery should be considered for injection therapy.

The procedure of injection of esophageal varices is not complicated. A No. 22 gauge stainless steel needle, soldered to the distal end of an ordinary suction tube, is the only special equipment necessary. On the proximal end of the suction tube, a small length of narrow rubber tubing is connected to the syringe with the 5 per cent sodium morrhuate solution. The system is first completely filled with the solution so that an exact knowledge of the amount of sodium morrhuate used is known. About 1 to 2 cc. of fluid is injected into each of several varices at one endoscopic examination. The procedure is repeated in several days as indicated.

During episodes of uncontrollable, active bleeding, esophagoscopy may be performed as an emergency diagnostic or therapeutic procedure. Supportive therapy cannot always be relied upon to carry the patient through until hemorrhage ceases spontaneously.

Tamponade for the immediate control of esophageal bleeding has proved relatively effective in some instances. The Sengstaken tube provides occluding pressure against the veins of the gastric cardia as well as those of the lower half of the esophagus. This consists of a triple-lumen tube, with the larger central channel used for gastric suction. The other two are used for inflation of the small spheroid gastric balloon and the larger cylindric esophageal balloon. The tube is swallowed and introduced into the stomach. After the distal cardiac balloon has been inflated with about 100 cc. of air, the tube is withdrawn until impingement of the cardiac balloon occurs. The esophageal balloon is now inflated to obtain even pressure against the esophageal varices, which maneuver also prevents dislodgement of the tube.

It is suggested that esophagoscopic examination of any patient with unexplained hemorrhage from the upper gastrointestinal tract become a routine procedure, to exclude esophageal varices. If bleeding varices are encountered, the hemorrhage may be temporarily controlled by pneumatic pressure with a balloon.

The problem becomes more pressing if the hemorrhage persists in spite of pneumatic pressure or recurs after withdrawal of the Sengstaken tube. The tube may be allowed to remain *in situ* for two to three days, during which time frequent aspiration of stomach contents must be performed. If the hemorrhage is uncontrolled after pneumatic pressure and supportive therapy, direct examination for the source of bleeding becomes imperative. The larynx should be intubated with a cuff, preferably under general anesthesia, in order to prevent spill-over of blood into the trachea. An esophagoscope is then inserted, with the intra-

tracheal tube *in situ*, and the contents of the esophagus are aspirated. If no varices of the esophagus are encountered, it can be stated that the bleeding is not esophageal in origin and that a gastric lesion probably exists.

BENIGN TUMORS OF THE ESOPHAGUS

Non-malignant tumors of the esophagus are indeed rare and are almost invariably non-epithelial in origin. Benign tumors may be classified as two types: (1) the intraluminal, including the fibrolipoma and fibromyxoma; and (2) the intramural, comprising the various leiomyomas.

The fibrolipomas most often arise at the opening of the esophagus by a limited attachment or pedicle and become suspended in the lumen of the upper esophagus. They may attain a rather large size, 7 to 10 cm. in length, but are always covered by intact mucosa. They have been known to become dislodged into the pharynx following an eructation and obstruct the laryngeal vestibule causing severe dyspnea.

On hypopharyngoscopy, a round smooth mass covered by intact mucosa may be seen attached to the postcricoid region. The tumor is freely movable and can easily be grasped and displaced into the pharynx. The best method of removal is by suspension laryngoscopy. By this procedure an excellent exposure is obtained and both hands are rendered free to extirpate the neoplasm by dissection and coagulating snare. In a recent case, a fibrolipoma was located in the lower half of the esophagus.

The intramural leiomyomas are more often located in the lower third of the esophagus. In a few instances the myomas also extend into the stomach. These tumors are not pedunculated. They have a firm consistency and are always covered by intact mucosa. They appear as oval or round submucosal masses which occasionally encircle the esophagus in the form of a U.

On endoscopy, the lumen of the esophagus seems to be compromised by an extra-esophageal mass. Occasionally the tube cannot be passed beyond the proximal bulge of the tumor. More often, with diligent care the main lumen of the esophagus can be recognized at an acute angulation. The tumor mass can be displaced by the esophagoscope and the remainder of the esophagus visualized. These intramural tumors are always covered by an intact mucosa. When the diagnosis of a leiomyoma is considered by the roentgenologist and the findings on endoscopy, as previously described, support this view, a biopsy is not necessary. As a matter of fact, a biopsy will not be satisfactory unless taken deep to the mucosa. The resultant ulceration of the mucosa may render subsequent enucleation of the tumor by the surgeon more difficult. Should ulceration of the mucosa be present at the initial esophagoscopy, the likelihood of malignancy or leiomyosarcoma exists and a biopsy is indicated.

MALIGNANT TUMORS OF THE ESOPHAGUS

Further progress in the study of cancer of the esophagus will follow the development of improved methods for establishing earlier diagnosis.

Two essential methods are utilized at the present time to establish the diagnosis of carcinoma of the esophagus, namely, roentgenography and endoscopy. There is little doubt that the earliest lesion can best be diagnosed by endoscopy. It should be remembered however, that in the majority of instances only the most proximal portion of the esophageal carcinoma can be seen by the endoscopist. The full limits of extension of the tumor are best determined by x-ray.

Carcinoma occasionally develops in the presence of benign lesions of the esophagus such as stricture, cardiospasm, peptic ulcer and hiatal hernia. An adequate biopsy will indicate the diagnosis.

Carcinoma of the upper esophagus most frequently occurs in the postcricoid region and at the mouth of the gullet. The incidence of postcricoid carcinoma is far greater in women than men (a ratio of 8:1). There seems to be little doubt that carcinoma in this location is definitely related to the so-called Plummer-Vinson syndrome. Chronic hypopharyngitis seems to be the precursor of this malignant change.

The history of progressive dysphagia accompanied by discomfort or pain in the ear on swallowing is suggestive of a lesion at the postcricoid region.

Indirect laryngoscopy usually reveals a normal larynx. In advanced cases, the upward extension of the neoplasm may be visible in the depths of the pyriform fossa or an unexplained vocal cord paralysis may be detected.

Direct visualization by esophagoscopy is essential for diagnosis. The lesion may be encountered just above the cricopharyngeus in the postcricoid region where ulceration or nodular thickening may be seen. Often the sphincter at the cricopharyngeus is visualized but the esophagoscopic tube cannot be passed because of the infiltration and stenosis. A small bougie might be accommodated but no attempt should be made to force the esophagoscope through the stenosis lest a perforation result.

During the past several years, ten cases of postcricoid carcinoma have been observed in which the evidence seemed to suggest that the lesions developed subsequent to radiotherapy administered fifteen to twenty-five years previously. These patients had been treated by radiation either for hyperthyroidism, tuberculous lymph nodes or carcinoma of the vocal cord. There is considerable evidence to substantiate the opinion that malignant transformation of mucosal surface can occur after radiation therapy.

Carcinoma of the middle or lower third of the esophagus may present the same endoscopic appearance. The x-ray film usually discloses the site of the suspected lesion. On esophagoscopy some inflammatory or hyperplastic mucosal changes may be seen proximal to the tumor. Care should be exercised to introduce the esophagoscope beyond this area until the lesion itself is identified, otherwise the biopsy may not include the neoplastic tissue.

The tumor may present itself as an ulcerative, polypoid or infiltrative lesion usually producing some obstruction. Occasionally, with large polypoid tumors of the middle third of the esophagus, the esophagoscope may be passed beyond the distal limit of the neoplasm.

The earliest squamous cell carcinoma encountered was 2 cm. in length and surrounded by normal esophageal mucosa on all sides. The patient is alive nineteen years after resection.

The lower third of the esophagus is the most common site for carcinoma of the esophagus. In this location the malignant tumor must be differentiated from marginal ulceration, achalasia and peptic stenosis.

Adenocarcinoma of the cardia may extend proximally to involve the lower esophagus. Such lesions frequently produce organic stenosis of the lower esophagus by submucosal infiltration. The overlying mucosa appears puckered or nodular and the lumen becomes conically narrower with the tapered end toward the stomach. The rosette may appear infiltrated and will not yield to the pressure of the distal end of the esophagoscope. Such resistance to passage may differentiate malignant infiltration of the sphincteric canal with proximal dilatation of the esophagus from dilatation due to true cardiospasm. A specimen from the narrowed lumen of the rosette or a deep biopsy may show submucosal adenocarcinoma extending from the cardia. A microscopic diagnosis of adenocarcinoma almost invariably indicates a neoplasm of the cardia except in the very rare instance of primary adenocarcinoma of the esophagus. In the latter, the lesion originating from submucosal esophageal glands must be completely surrounded by esophageal mucosa in order to be considered primary. The cardia must be free of neoplasm.

It should be stressed that a single negative biopsy may not be sufficient. When the clinical and roentgen evidence strongly suggest malignancy, the examination and biopsy should be repeated. This is especially true of cancer of the cardia.

In advanced carcinoma of the esophagus with malnutrition, it may be necessary to pass a Levin tube for alimentation, especially during the administration of radiotherapy. In such instances, the stenosed segment might be traversed with a narrow esophagoscope and then the rubber or plastic feeding tube can be fed through the esophagoscope into the stomach.

Rarely, other malignant neoplasms involve the esophagus and may be diagnosed by biopsy. Recently a polypoid primary malignant melanoma was encountered and subsequently successfully resected. Lymphosarcomatous invasion of the stomach produces prominent greyish rugae from which a biopsy may prove the diagnosis. Hodgkin's disease may invade the esophageal wall and elevate the mucosa to permit the taking of a biopsy specimen.

In scleroderma and occasionally in dermatomyositis a smooth narrowing of the lower esophagus may be encountered with atrophic mucosa and a rigid wall. If the collagen disease has involved the sphincter mechanism at the lower end of the esophagus, peptic reflux with marginal ulceration may be present.

Finally, all patients with persistent dysphagia not explained on a neurologic basis should undergo esophagoscopy.

Congenital Anomalies of the Esophagus

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CONGENITAL anomalies of the esophagus result from a fault in development at about the fourth week in the life of the embryo. A groove in the ventral wall of the primitive pharynx deepens and converts into the laryngotracheal tube. This tube is in intimate relation to the upper part of the foregut, the lengthening of which forms the esophagus. Incomplete separation of the trachea and esophagus, and failure of recanalization of the esophagus result in: (1) atresia of the esophagus with tracheoesophageal fistula, (2) atresia without tracheoesophageal fistula, and (3) tracheoesophageal fistula without atresia. Other anomalies of the esophagus are: (1) reduplications of the esophagus, (2) paraesophageal cysts, and (3) diverticula.

Atresia of the esophagus with or without tracheoesophageal fistula is the most common anomaly of the esophagus. Humphreys et al. [1] report that it occurs in a ratio of approximately 1:3,000 births. Its surgical correction is a difficult task, which taxes the skill and experience of the surgeon, anesthetist and hospital staff involved in the postoperative care. The reader is referred to excellent contributions that have been made by Lanman [2], Ladd and Gross [3], Leven [4], Haight [5], Swenson [6], Gross [7], Humphreys et al. [1], Potts [8] and others. Unless corrected surgically, the anomaly is incompatible with life since the infant cannot be fed. The early threat, however, is chiefly to the respiratory tract from aspiration of secretions from the mouth and reflux of the gastrointestinal secretions through the lower esophagus into the trachea. This latter danger is the greater one. During the period before the anomaly is corrected the infant should be placed in a semi-upright position to allow gravity to overcome the tendency toward reflux. Nursing attendants should be vigilant in the aspiration of pharyngeal secretions. There should be no immediate concern about the giving of parenteral fluids, since the newborn infant at term is plethoric and well able to endure three or four days without fluids.

DIAGNOSIS

Atresia of the esophagus should be suspected when a newborn infant drools excessively, chokes upon feedings and has cyanotic episodes. The diagnosis can be confirmed by the inability to pass a catheter into the stomach and the visualization of a blind upper pouch on roentgenogram after putting opaque media (never barium) into the upper esophagus. The presence of air in the stomach and intestines as shown by the roentgenogram establishes the fact that a tracheoesophageal fistula is present. The absence of air suggests that there is no fistula between the lower esophagus and trachea but does not rule out this possibility. Tracheoesophageal fistula without atresia should be suspected when a newborn infant has severe coughing episodes when fed. The fistula may be demonstrated by roentgenograms taken after opaque media has been placed in the esophagus with the infant in the prone position.

ANATOMY OF THE ANOMALIES

Atresia of the esophagus with tracheoesophageal fistula, in which the lower esophagus enters the posterior wall of the trachea, makes

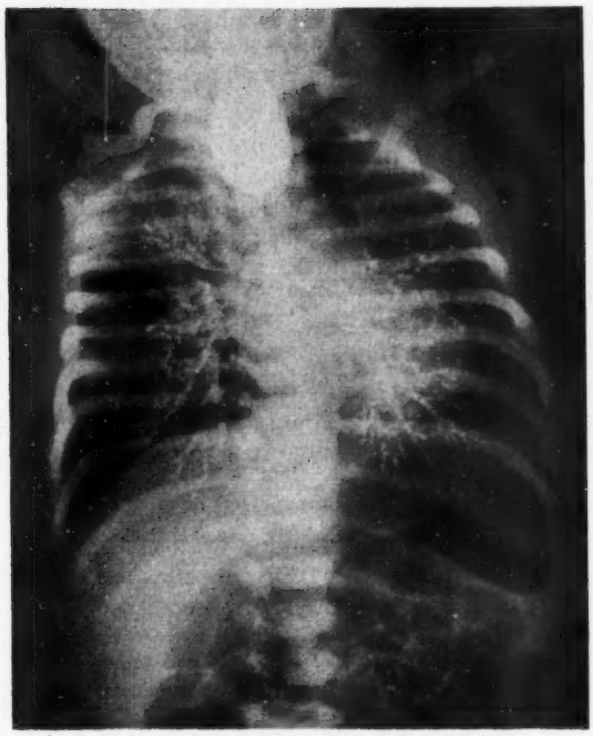

FIG. 1A. This roentgenogram shows the typical anatomy of congenital atresia of the esophagus with tracheoesophageal fistula. Note small tapered lower segment communicating with trachea. (It is not advisable to use this amount of opaque media during the roentgenographic examination.)

FIG. 1B. This is the same type of anomaly as shown in Figure 1A. Note long upper blind pouch overlapping large lower segment which forms a fistula with the trachea.

up 90 per cent of the anomalies of this type. There is considerable anatomic variation in this anomaly. The upper esophagus, which is a blind pouch, is uniformly thickened but may vary in length from a small pouch high in the thorax to a blind segment extending almost to the diaphragm. (Fig. 1.) In like manner, the lower esophagus may be well developed and enter the posterior tracheal wall with a patulous opening as high as 2 cm. above the bifurcation of the trachea, or it may be a small underdeveloped muscular tube which tapers sharply at its upper end to form a small fistula at the bifurcation of the trachea or at the upper portion of the left main bronchus. Thus the two esophageal segments may overlap 2 to 3 cm. or there may be a gap of similar length between their two ends. When an overlapping esophagus is exposed at the time of surgery, it may appear normal externally. However, the upper pouch will be seen to bulge when pressure is exerted on the anesthetic bag, thus confirming the atresia which has been demonstrated on roentgenograms.

Atresia of the esophagus without tracheoesophageal fistula is variable in the length of the upper esophageal pouch. (Fig. 2.) The lower esophagus may vary from an atretic cord of tissue to a sharply tapering esophagus ending blindly in the posterior mediastinum adherent to but not communicating with the trachea. A variant of this anomaly is the congenital esophageal web located at the mid-esophagus.

Tracheoesophageal fistula without atresia is represented by a well developed esophagus which is intimately adherent to the trachealis muscle along the lower portion of the trachea with a fistulous opening above the bifurcation.

In rare instances two fistulas may be present, with the upper segment as well as the lower segment communicating with the trachea.

SURGICAL APPROACH

We prefer the extrapleural over the intrapleural approach to the posterior mediastinum for the surgical correction of these anomalies. This approach is no more time-consuming, minimizes the anesthetic problem and yields a quieter mediastinum during dissection and

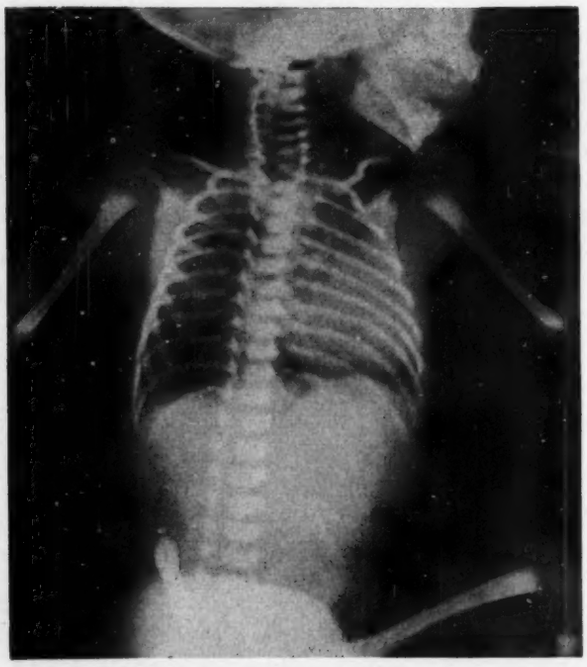

FIG. 2. Roentgenogram of infant with atresia of esophagus without tracheoesophageal fistula. Note absence of air in stomach and intestines.

the performance of the anastomosis. Most important, however, this approach makes it possible to localize any infection that may occur due to a leak of the esophagus at the point of anastomosis.

The infant is placed on his left side with the right arm drawn forward. A 4 cm. incision parallel to the vertebral border of the scapula is made in the skin and subcutaneous tissue of the right side of the chest. The fascia and vessels of the auscultatory triangle are then incised. Two small retractors are placed in the opening and the space is enlarged by traction. It is seldom necessary to cut into the muscles of the chest wall. This maneuver reduces blood loss and operative time. A long length of the fourth rib is removed subperiosteally. An incision is then made in the periosteal bed and dissection started in the extrapleural plane. We have found that a small blunt dural elevator is an excellent instrument for accomplishing this dissection. As the extrapleural space is developed, the ribs are gradually spread by a small self-retaining retractor. In developing the extrapleural space care should be exercised in stripping the pleura from the region of the head of the ribs where it is most adherent. The azygos vein should be doubly ligated and divided in order to gain freer access to the site of the fistula. The upper esophageal segment is usually readily identified as soon as the upper mediastinum is exposed. The anesthetist can assist in its identification by passing a small catheter into the esophagus. A suture is placed in its lower tip for traction. The upper esophagus is then mobilized well up into the dome of the chest using blunt and sharp dissection. This segment is usually intimately connected to the posterior wall of the trachea at its lower portion. The dissection is facilitated by first separating the esophagus from the trachea high in the chest and gradually working in a downward direction. By keeping the esophagus under tension, the plane of separation can be followed and an inadvertent opening into the tracheal lumen avoided. Complete mobilization of the upper segment is most important if there is a gap to be bridged in performing the anastomosis, since it is hypertrophic, has good blood supply and lends itself well to lengthening. The lower esophagus is next identified. When it is not found immediately, the operator can locate it by following the right vagus nerve. The lower esophagus should be encircled by a ligature so that gentle traction will permit easy dissection up to the fistulous opening with the trachea. It is then severed, leaving sufficient tissue to permit closure of the opening into the trachea. This closure should be made with fine sutures in a transverse direction so as to reduce to a minimum the mucosal recess remaining within the tracheal lumen. In the dissection and subsequent anastomosis the esophageal segments should be handled as little as possible with grasping instruments to avoid injury to the delicate tissue.

Anastomosis. An uncomplicated successful result in the surgical correction of atresia of the esophagus with tracheoesophageal fistula depends on the healing of the reconstructed esophagus. Great care must be taken in performing the anastomosis. If the lower segment is small and the tissue layers are delicate or have poor blood supply, it is advisable to free more of the lower esophagus from its bed so that healthier, more substantial tissue is available for the anastomosis. Even though the gap is made greater, it may be necessary to resect a portion of the tip of the lower esophagus. It is best to sacrifice this tip after a posterior row of fine silk sutures has been placed to approximate the musculature of the lower segment to the tip of the upper segment at the desired

point of anastomosis. After these sutures have been tied, the excess amount of tissue is trimmed away from the lower segment. The musculature of the lower tip of the upper segment is then excised and the mucosal layer opened. Accurate closure of the mucosal layer is carried out using fine silk sutures on atraumatic® needles, inverting the knots into the lumen. Before the final sutures are tied, a small plastic tube (No. 190) may be passed down through a nostril into the pharynx and upper esophagus and on through the anastomosis into the stomach, as advised by Garrison and Mayer [9]. This tube will be used for feeding the infant in the immediate postoperative period. Following completion of the mucosal layer, interrupted sutures of the same material are used to approximate the muscular layer, avoiding mattress sutures since they tend to impair the blood supply of the ends of the segments. Whenever possible the anastomosis should not be in juxtaposition to the repair of the fistula in the posterior wall of the trachea. If readily available, a layer of mediastinal fascia should be placed between the trachea and esophagus in an attempt to prevent recurrence of a tracheoesophageal fistula in the event of leakage at the site of anastomosis. If the esophagus is under any tension following the anastomosis, the upper segment should be secured to the fascia overlying the bodies of the vertebrae with fine sutures so as to relieve tension on the anastomosis when the infant swallows, as suggested by Swenson [6]. We have employed drainage of the mediastinum using a small Penrose drain. Others have questioned this practice, but we believe it has value in giving daily observation of the character of the drainage. The earliest indication of a leak is the appearance of saliva and mucus on the dressing. This finding makes immediate gastrostomy mandatory.

In general the surgical approach for correction of tracheoesophageal fistula without atresia and atresia without fistula does not differ from that for atresia with fistula. Each anomaly presents its own technical problem. In the case of the fistula without atresia the major problem is the separation of the esophagus from the trachea to which it may be fused for a distance of approximately 1 cm. above and below the fistula. This may make it necessary to transect the esophagus, close the fistula and perform an end-to-end anastomosis. Congenital esophageal webs, which are a form of atresia without fistula, usually require resection of the segment bearing the web and end-to-end anastomosis. The use of a longitudinal incision in the esophagus through the web and transverse repair results in stricture formation at the site of the repair. Occasionally the web will rupture spontaneously, permitting feedings through a narrow lumen. We discovered that this had happened in a two year old child who swallowed a nickel which lodged in the mid-esophagus. When the coin was removed at esophagoscopy, the partially obstructing web was seen. At a later esophagoscopy this web was cut, allowing restoration of an almost normal lumen.

Anesthesia. Our present preference is ether-oxygen anesthesia administered with a tight-fitting mask. Local anesthesia alone was used in some of our cases, but its advantages seem to have been more than outweighed by the difficulties caused by a struggling infant. In our experience intratracheal intubation led to too many complications associated with subglottic edema and obstruction of the upper air passages, which in two instances necessitated tracheostomy. Use of the extrapleural approach lessens the need for positive pressure during anesthesia.

Gastrostomy. Gastrostomy is a useful procedure in the pre- and postoperative management of congenital atresia. The simple Stamm type gastrostomy as described by Gross [7] is relatively safe to perform, is adequate for infant feeding and closes quickly when the tube is removed. In the infant with atresia without fistula the gastrostomy may be used immediately for feeding purposes. Gastrostomy should be performed as a decompression measure in atresia with fistula in which severe distention of the stomach and intestines causes respiratory embarrassment. The gastrostomy tube should be left open or kept under gentle suction but should not be used for feeding purposes until the fistula is closed. This measure is especially valuable when transporting an infant to a distant city for definitive care.

Gastrostomy may be performed immediately following the surgical repair of the anomaly if the infant's condition seems satisfactory, or it may be performed as a separate procedure one or more days later. In patients having excellent anastomosis and being fed through a small plastic tube, gastrostomy will not be necessary.

Gastrostomy once instituted should be maintained even though not used until it is evident that feeding by mouth will be satisfactory and that dilatation of a stricture at the site of the anastomosis will not be required.

Administration of Fluid. Administration of fluid will not be necessary during the first three or four days of life since the full term newborn is plethoric. If, however, the diagnosis has been delayed and the infant is dehydrated, fluids should be given subcutaneously. During the surgical procedure 10 cc. of whole blood per pound of body weight is given through a cannulated ankle vein. An additional 10 cc. of clear fluid per pound of body weight is given slowly at the conclusion of the operation if the infant is dehydrated. The cannula is then removed to eliminate the danger of fluid overload, which is especially hazardous for the infant in respiratory distress. Following surgery, daily fluids are limited to 50 cc. per pound of body weight for at least three days. The fluids are given through the small indwelling plastic tube or the gastrostomy tube. Beginning with the fourth postoperative day the amount of fluid is gradually increased until the child is receiving an adequate intake.

A roentgenogram is made of the esophagus, using opaque media, on the eighth postoperative day if there has been no evidence of leakage from the anastomosis. Feedings by mouth, using an eye dropper as suggested by Potts [8], are begun if this examination reveals a well healed esophagus. Gastrostomy feedings are continued if a leak at the anastomotic site is demonstrated. Most of these leaks demonstrated at this time are small and will heal with the esophagus at rest and with adequate drainage of the mediastinum.

Tracheostomy. Tracheostomy may become necessary in the management of the infant having tracheoesophageal fistula because of obstruction of the upper air passages due to trauma from instrumentation, and to provide an avenue for aspiration of bronchial secretions to help clear severe pneumonitis caused by aspirational episodes. Care of the tracheostomy in the newborn is difficult and requires constant nursing attention. Decannulation often must be delayed.

Complications. The early complications are atelectasis and pneumonitis due to the dual threat to the respiratory tract. Antimicrobial therapy employing penicillin and streptomycin should be started immediately. Atelectasis of a single lobe, usually the right upper lobe, is not a serious complication and often clears during the operation. Overloading the infant in respiratory distress with parenteral fluids should be studiously avoided.

Late complications are almost always due to faults in healing at the site of anastomosis, namely, (1) esophagocutaneous fistula, (2) recurrent tracheoesophageal fistula, and (3) stricture. The esophagocutaneous fistula will close if adequate drainage of the mediastinum is maintained. This complication, however, does predispose to stricture formation. Recurrence of the tracheoesophageal fistula should be suspected when, following surgical repair, an infant coughs excessively, especially when fed. This complication frequently is accompanied with a stricture at the anastomotic site and requires reoperation as soon as the mediastinitis has completely subsided. Some degree of stricture formation must be expected in all infants having repair of atresia of the esophagus. (Fig. 3.) In the early postoperative period no attempt should be made to dilate the stricture since it is very easy to tear the fragile tissue. Gastrostomy feeding should be continued and secretions collecting in the pharynx and upper esophagus aspirated. In many infants, as swelling at the anastomotic site subsides, an adequate lumen will result. In others, when the esophagus is well healed, one or two careful dilatations may be all that is required. We prefer to treat high grade strictures by reanastomosis following excision of the narrow esophageal segment.

Substitution Procedures. Although primary anastomosis of the esophageal segments after division and closure of the fistula is the procedure of choice for atresia of the esophagus with tracheoesophageal fistula, this procedure is not applicable in the absence of an adequate lower esophageal segment or if the gap between the upper and lower segments is too great to permit satisfactory anastomosis. In these patients substitution procedures, using either stomach or jejunum, are necessary to provide alimentary continuity. The high operative mortality of an unsatisfactory end-to-end anastomosis may be reduced by division and closure of the fistula, cervical esophagostomy and gastrostomy at the first operation, to be followed at a later date with a substitution procedure. Many different types of reconstructive procedures have been

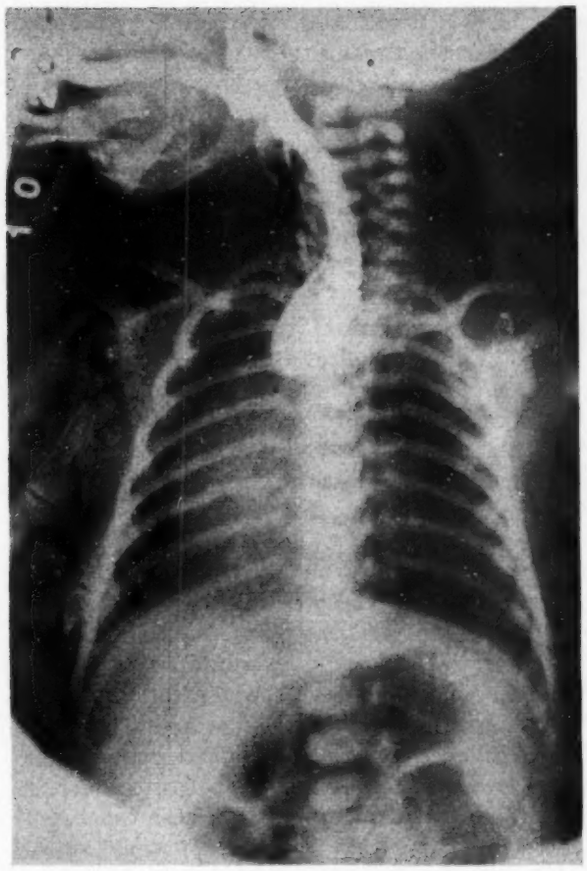

FIG. 3A. Infant with atresia of the esophagus with tracheoesophageal fistula.

FIG. 3B. Same infant ten days following closure of fistula with anastomosis of esophagus. Note narrowing at the site of anastomosis. This child now swallows a normal diet without difficulty.

described for the treatment of anomalies unsuitable for anastomosis. Gross [7] has suggested sliding the stomach and esophagus through a dilated hiatus in the diaphragm to facilitate an anastomosis between a short lower esophageal segment and the upper esophageal pouch. Byron [10], Swenson [11] and others have performed immediate transthoracic esophagogastrostomy in the newborn. The mortality of primary intrathoracic esophagogastrostomy has been high. In general, those who survived have failed to mature physically at a normal rate. Sweet [12], Haight [5] and Potts [8] have all emphasized the hazards of intrathoracic placement of the stomach in a newborn infant whose respiratory function has already been impaired by the anomaly itself. Delayed procedures for restoration of alimentary continuity would appear to be prudent where primary anastomosis is impossible or unwise.

The absence of air in the stomach or intestine is not sufficient indication to abandon extrapleural mediastinal exploration. Atresia of the esophagus without the fistula but with the lower esophageal segment in muscular continuity with the upper pouch was found in one of our patients. Secondly, mediastinal exploration is necessary since it is possible to have a small fistulous opening which would otherwise not become apparent until feedings through the gastrostomy are attempted.

Alimentary continuity may be provided by elevation of either stomach or jejunum to the neck for anastomosis to the esophagus. In general, most surgeons have placed the organ being substituted on the anterior chest wall outside the thorax. Sweet [12] successfully transplanted the stomach of a twenty-one month old child into the chest and anastomosed the fundus to the cervical esophagus. Rienhoff [13], Bigger [14] and Potts [8] placed the entire stomach beneath the skin of the anterior chest wall and performed cervical esophagogastrostomy. Potts subsequently transplanted the stomach into

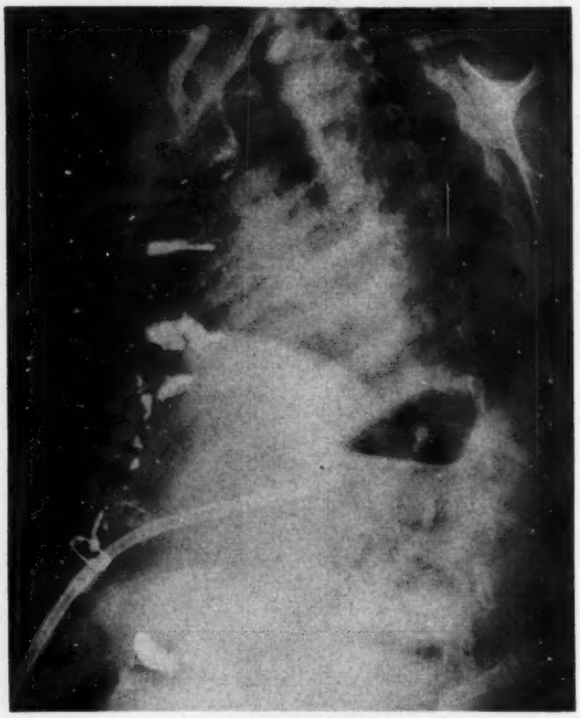

FIG. 4. Substernal transplantation of the jejunum and cervical esophagogastrostomy in a child in whom end-to-end anastomosis was impossible.

the left side of the chest because of the undesirable cosmetic appearance. Others, including Davis and Stafford [*15*], Ladd and Swenson [*16*] and Lam [*17*], have used a segment of jejunum and a skin tube. Longmire [*18*], Leven and Varco [*19*], and others have used a limb of jejunum as an antethoracic transplant similar to the method of Roux [*20*] and Yudin [*21*]. Cervical esophagojejunostomy is completed at a later stage.

The disadvantages of an intrathoracic stomach in a child and the undesirable cosmetic appearance of an antethoracic stomach or jejunal loop make attractive intrathoracic, substernal placement of a Roux-Y jejunal loop as originally suggested by Robertson and Sarjeant [*22*]. The substernal route constitutes the shortest distance to the neck and it is extrapleural, obviating the risks of an intrapleural placement.

We have utilized substernal placement of the jejunum and cervical esophagojejunostomy with satisfaction in three children in whom primary end-to-end anastomosis of the esophageal segments was impossible or unsatisfactory. (Fig. 4.) In all patients cervical esophagostomy, division and suture of the fistula if present, and gastrostomy were performed at the time of the first procedure. These children were fed by gastrostomy tube for nearly two years, at which time a Roux-Y loop of jejunum was prepared and placed in a substernal tunnel for anastomosis with the proximal esophageal segment in the neck.

The crux of the operation consists of careful preparation and straightening of the jejunal loop to be transplanted. After inspection and study of the jejunal mesenteric vessels, the serosa, fat, lymphatics and nerves are carefully stripped from these vessels well up onto the jejunum. In accordance with the vascular pattern, two or three principal jejunal vessels are clamped, taking care to preserve an adequate blood supply for the proximal jejunal segment just below the ligament of Treitz. After determining the adequacy of the blood supply and the desired length of the jejunal loop the appropriate principal vessels are divided and ligated. The jejunum is divided 6 to 10 cm. beyond the ligament of Treitz and the length of the resulting jejunal segment is measured. Division of additional main jejunal vessels may be necessary to provide sufficient length to reach the neck. However, it is important to realize that the more jejunal vessels that are divided, the greater is the distance necessary to be bridged by the straightened jejunal segment.

The substernal space is entered in the extraperitoneal and extrafascial space, freeing the diaphragm widely from its attachments to the sternum and costal margin. By blunt dissection with the fingers the anterior mediastinal space is widened, pushing the pleura to either side, progressing gently in an upward direction anterior to the pericardium.

A cervical incision is made along the anterior border of the sternocleidomastoid muscle and the proximal esophagus dissected free. The deep cervical fascia is incised and the sternothyroid and sternohyoid muscles are divided. The substernal space is then entered from above and the tunnel widened to admit two or three fingers. By blunt dissection from above and below, the tunnel is extended in both directions until the fingers meet. Attention should be directed to the preparation of a wide tunnel to provide ample space for the transplanted jejunum.

The prepared loop of jejunum, having been previously temporarily closed and the sutures

left long, is then brought through the transverse mesocolon, anterior to the stomach and out of the peritoneal cavity into the substernal space. It is both pulled and gently pushed through the anterior mediastinal substernal tunnel to the neck. Great care is taken to avoid tension on blood vessels and in general to allow the jejunum to lie on its side, usually toward the right. If sufficient length of jejunum has been provided, end-to-end esophagojejunostomy is performed. The manubrium may be divided and anastomosis performed beneath it if necessary.

After the completion of the cervical procedures end-to-end anastomosis of the proximal jejunum to the side of the straightened loop below the transverse mesocolon is accomplished. The mesocolon is sutured to the jejunum and the abdominal incision closed about the bowel as it leaves the peritoneum. No attempt should be made to anastomose the stomach and transplanted loop of jejunum at this stage. These children usually require gastrostomy feeding for a variable period of time during the postoperative period and until such time as the child has become educated in oral feeding. At a later date side-to-side anastomosis of the jejunum to the antrum of the stomach may be easily accomplished to correct the by-pass of the stomach and its deleterious effects on development.

RESULTS

The salvage rate for atresia of the esophagus corrected surgically is steadily improving since the first successful end-to-end anastomosis performed by Haight in 1940. Reports by Gross [7], Haight [5], Humphreys et al. [1], Swenson [6], Potts [8] and others show a salvage rate ranging from 40 to 50 per cent of infants treated for this anomaly since 1939. In the later years of this period the salvage rate in many series of cases has reached as high as 80 per cent. We have twenty-seven living children from a group of sixty-five babies (41.5 per cent) treated since 1938. During the past five years our results have improved with the salvage of twenty-two of thirty-four babies treated (65 per cent). Other associated anomalies incompatible with life and the poor general condition of many of these babies when presented for surgical therapy make it difficult to improve these results appreciably.

OTHER ANOMALIES

Reduplications of the esophagus and paraesophageal cysts may not be symptom producing. Often their presence is indicated by an abnormal shadow seen on a roentgenogram of the chest. Their surgical removal usually can be easily accomplished by employing a transpleural approach to the posterior mediastinum. Epiphrenic esophageal diverticula are considered by most authors to be acquired diverticula of the pulsion type. One patient treated surgically by us for this type of diverticulum had one brother who also had a similar diverticulum removed surgically and another brother who was the father of two infants, both born with atresia of the esophagus. Our experience with this family and the history often obtained of symptoms going back to early childhood lead us to believe that these diverticula may be congenital in origin. They are easily excised surgically using a transpleural approach.

CONCLUSION

Congenital anomalies of the esophagus in which atresia or a fistulous connection are present are urgent surgical problems. More than half of these infants can be salvaged by direct end-to-end anastomosis of these two segments of the esophagus or by delayed substitution procedures when the former is not feasible. Associated anomalies incompatible with life, aspirational episodes causing pulmonary infections and complications due to faulty healing of the anastomosed esophagus are the greatest obstacles encountered in the surgical management of these infants. Other esophageal anomalies present surgical problems less urgent in nature and more easily managed.

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Pharyngoesophageal Diverticulum

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PHARYNGOESOPHAGEAL diverticulum is not a common disease but the relentless progress of the symptomatology associated with this lesion leads almost inevitably to surgical intervention. The continuing controversy as to the most appropriate operative procedure to be employed in the management of this condition coupled with the ultimate necessity for surgical intervention makes this condition a lively subject for surgical discussion.

For many years Lahey had an avid interest in this problem and was an ardent exponent of the two-stage operation for the excision of these diverticula. It seems worth while, therefore, in a symposium on the esophagus and its diseases to record once more the cumulative experience at the clinic in the management of this problem.

The detailed historical background of pulsion diverticula of the pharyngoesophagus was reviewed by Lahey and Warren in an article published in 1954 [1], and for this reason only limited reference will be made to these historical data in this current discussion.

ETIOLOGY

The precise etiology of pulsion diverticula of the pharyngoesophagus is unknown. Certain physiologic and anatomic factors appear so unquestionably to be involved in the genesis of these diverticula that some consideration should be given to this phase of the subject.

The simple act of swallowing plays such an important part in the production of pharyngoesophageal diverticula that any discussion of etiology should perhaps begin with an analysis of this phenomenon. The comprehensive description of Negus [2] and the discussion of the act of swallowing from the standpoint of the endoscopist by Jackson and Jackson [3] contain most of the basic knowledge referable to this physiologic activity. It is believed that a peristaltic wave is initiated by the pharyngeal constrictors beginning in the nasopharynx, passing downward and ending in the pharyngoesophageal junction. It is at this point in the act of swallowing that undue pressure may be exerted at the pharyngoesophageal junction. It is to be noted that, anatomically speaking, the lowest inferior constrictors and the cricopharyngeal fibers are disposed obliquely at the pharyngoesophageal junction, thus producing in a small triangular area a weak spot at this point. In the normal act of swallowing, as the peristaltic wave in the pharyngeal constrictors approaches the pharyngoesophageal junction, a coordinated relaxation of the cricopharyngeal sphincter should occur to permit the unobstructed propulsion by the constrictors of the mass of food into the esophagus. If this coordination involving the relaxation of the cricopharyngeal sphincter during this initial peristaltic wave of the pharyngeal constrictors is disturbed, undue pressure will be exerted at the weak point previously described. Associated with failure of the coordinated relaxation at the pharyngoesophagus, a small bulge occurs at the junction of the pharynx with the esophagus on the posterior wall which eventually will be the beginning of an esophageal diverticulum.

After the initial establishment of a bulge at the junction of the pharynx and the esophagus, the ultimate development of the pharyngoesophageal pulsion diverticulum is merely a matter of repetition of this obstruction during

the descent of the bolus of food until a true sac consisting of mucosa and submucosa of the pharynx attains sufficient size to hold food and saliva. Subsequently, by weight of its own contents and by repeated distentions during the ingestion of food, it is capable of becoming larger and larger until it acquires the classic characteristics of a pharyngoesophageal diverticulum.

The true nature of the resistance to the passage of food through the pharyngoesophageal junction is unknown, but it has been speculated that this relative obstruction, as pointed out by Hoover of the Department of Otolaryngology at the clinic, may be due to spasm, muscular incoordination or increased tone of the cricopharyngeal muscle. Ultimately, fibrosis may occur and involve the mucous membrane, the submucosa and even the adjacent muscle.

DIAGNOSIS

The diagnosis of pharyngoesophageal pulsion diverticula and more particularly the demonstration of the size and the relative stage of development are dependent on roentgenologic studies following the oral administration of barium. The experience at the clinic indicates that it is unnecessary to employ esophagoscopy either as a diagnostic procedure or as an adjunct to the operative measures during the surgical treatment of this condition. Furthermore, experience with several patients referred to the clinic following ill-advised or inexpert endoscopic manipulation in the presence of pulsion diverticulum of the pharyngoesophagus emphasizes the dangers inherent in this type of manipulation in this lesion except in the hands of an experienced endoscopist thoroughly conversant with the potential dangers of endoscopic manipulation in the presence of these lesions.

Technical considerations in the roentgenologic study of a patient suspected of having an esophageal diverticulum should emphasize particularly the pitfalls of studying such a patient with anteroposterior roentgenograms alone. Since an esophageal web may closely simulate the globular type of esophageal dilatation so characteristic of the second stage pharyngoesophageal diverticulum as viewed in the anteroposterior roentgenogram, one should always insist that a lateral or oblique view of the diverticulum be obtained. In the lateral view there is a characteristic spill-over from the top of the diverticulum into the longitudinal esophagus itself, which eliminates an esophageal web as a source of the globular dilatation previously described. If the course of the barium is followed through the pharynx into the esophagus in the presence of a pulsion pharyngoesophageal diverticulum, it will be noted that the diverticulum fills first to its most dependent portion; after the entire diverticulum has filled there is a spill-over into the longitudinal esophagus. This observation distinguishes it as a true pharyngoesophageal diverticulum. As emphasized by Hoover, the examiner should also be wary of a roentgenogram taken in the anteroposterior view in the presence of unilateral or bilateral paralysis of the pharynx. It is once more possible only by a lateral or semi-lateral view of the pharyngoesophageal junction following the ingestion of barium to distinguish between this type of defect and a true diverticulum.

In terms of the roentgenologic appearance of diverticula of the pharyngoesophageal area, it should be pointed out that any irregularity in the contour of the diverticulum should be sufficient to indicate the possibility that the diverticulum may be the site of a malignant tumor. Upon two occasions at the clinic, such a malignant lesion has been recognized on roentgenograms and subsequently proved at the time of excision of the diverticulum.

A final word of warning with respect to roentgenologic studies in pharyngoesophageal diverticula should refer to the postoperative appearance of such roentgenograms following the ingestion of barium. Before it is assumed that a recurrent pulsion diverticulum is present, it should be demonstrated that the longitudinal esophagus fills from spill-over at the level of the neck of the defect. This feature is emphasized because the routine postoperative roentgenograms taken immediately or remotely after the removal of a diverticulum or some time later may show some shelving at the point at which the sac was amputated, and to the inexperienced observer this shelving may be interpreted as a recurrence of the sac.

STAGES IN THE DEVELOPMENT OF PHARYNGOESOPHAGEAL DIVERTICULUM AND THE RELATIONS TO SYMPTOMATOLOGY

In order to understand the varying symptoms of esophageal diverticula as well as to

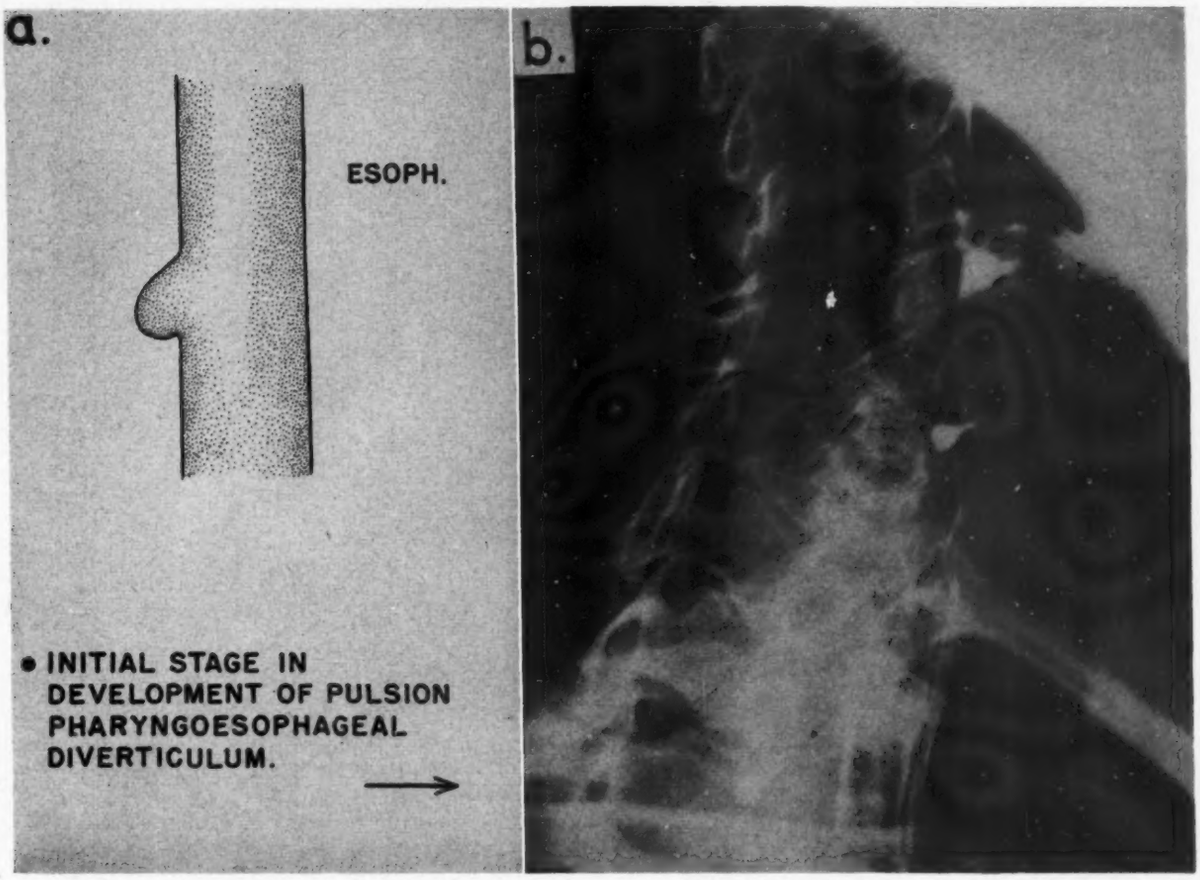

FIG. 1. (*a*) The first stage in the development of a pharyngoesophageal diverticulum is shown diagrammatically. In the roentgenogram (*b*) such a diverticulum is shown. (From: LAHEY, F. H. and WARREN, K. W. Esophageal diverticula. *Surg., Gynec. & Obst.*, 98: 1–28, 1954.)

appreciate the dangers of intrapharyngoesophageal manipulation and to apply properly certain surgical principles, one should bear in mind the three stages in the development of pharyngoesophageal diverticula which may be detected by roentgenologic, endoscopic and surgical evaluation of these lesions. These three stages, although arbitrary, are of practical significance. As the sac increases in size, it increases in length. Since the sac develops superior to the lowermost fibers of the cricopharyngeal muscle, the point of origin, and consequently the opening of the sac into the esophagus, remains in a fixed position even though the most dependent portion of the sac may descend ultimately into the mediastinum. The lateral expansion of the sac is limited by the large muscular structures of the neck and its posterior expansion is limited by the presence of the vertebral column. The only unobstructed direction in which the sac can enlarge is downward.

The first phase is shown diagrammatically in Figure 1*a*. At this stage no true sac exists but the initial bulge of the mucosa and submucosa through the triangular weak point at the pharyngoesophageal junction is evident. Symptoms at this stage are minimal and consist of occasional and temporary retention of food, usually of a dry or rough character. The patient may have a sense of a foreign body lodging at this point, with a reflex attempt to dislodge it by hawking. Although it is true that only a few diverticula will be seen at this stage of development, no attempt should be made to deal surgically with a diverticulum of this size and at this stage in its development if the symptoms are minimal. The opening into the diverticulum during the first stage of its development is lateral.

In the second stage of development a true sac, globular in shape, is evident on roentgenologic study with barium contrast media. The opening into the esophagus is still laterally

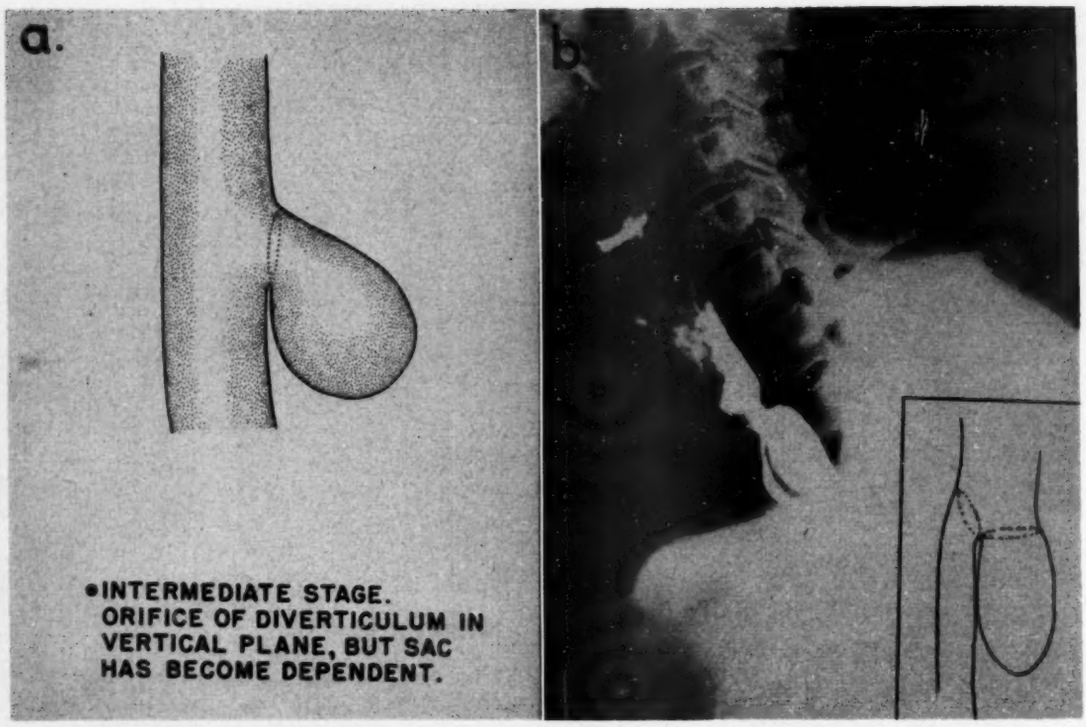

FIG. 2. (*a*) Diagrammatic representation of second stage in the development of pharyngoesophageal diverticulum. The opening into the diverticulum is still laterally disposed. (*b*) Gradual shifting of the axis of the esophagus in relation to the neck of the sac, with the opening into the diverticulum being converted into a transverse plane. The insert in (*b*) is a diagrammatic representation of third stage. (From: LAHEY, F. H. and WARREN, K. W. Esophageal diverticula. *Surg., Gynec. & Obst.*, 98: 1–28, 1954.)

placed and although some food will enter the diverticulum, most of the food continues by a direct pathway into the esophagus. (Figs. 2*a* and *b*.) The symptoms during this stage relate to the accumulation of food and liquid in this globular sac. These symptoms include occasional regurgitation of food taken at a previous meal. This regurgitation occurs quite frequently during the swallowing of another meal, between meals or while turning the head from side to side. A distressing feature of this manifestation of esophageal diverticula is the fact that the regurgitation may occur at night during sleep. Food and liquid may thereby be aspirated and be associated with severe paroxysms of coughing. This aspiration of regurgitated food has been responsible for a large number of these patients coming to the clinic for surgical relief. A few patients, after repeated aspirations, have had severe lung infections as well as lung abscesses.

The third phase in the development and symptomatology of pharyngoesophageal diverticula deserves particular attention. It is with the development of this stage that obstruction of the esophagus becomes increasingly manifest. The degree of obstruction may be sufficient to cause severe inanition. This inanition is due not only to the relative inability of the patient to swallow but also to the development of a fear of eating. The mechanics of obstruction of the esophagus caused by an esophageal diverticulum at this stage of development is shown in Figures 3*a* and *b*. In the roentgenogram (Fig. 3*b*) the mechanical explanation of the obstruction secondary to the diverticulum is well demonstrated. It will be noted that at this stage in development the opening into the esophagus distal to the diverticulum is now laterally disposed, while the opening into the diverticulum itself is transverse and in line with the long axis of the normal esophagus. This feature points up the danger of esophagoscopy or bouginage in this type of diverticulum.

The obstruction at this stage is not due, as one might think, to the pressure of the food-filled sac upon the esophagus but, by virtue of the weight of the sac and the shift of the axis

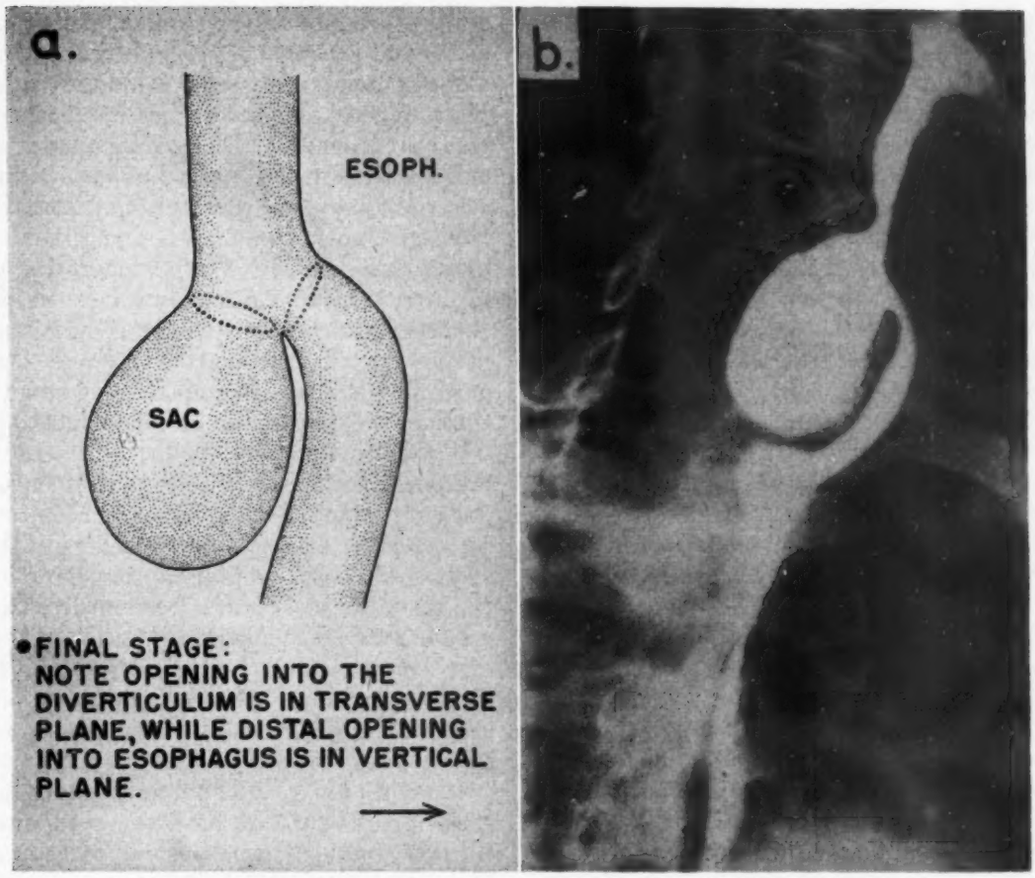
a.
ESOPH.
SAC
•FINAL STAGE:
NOTE OPENING INTO THE DIVERTICULUM IS IN TRANSVERSE PLANE, WHILE DISTAL OPENING INTO ESOPHAGUS IS IN VERTICAL PLANE.
b.

FIG. 3. (*a*) This diagram illustrates the conversion of the opening into the diverticulum from its original position on the lateral wall of the esophagus to the transverse plane. The opening into the true esophagus is now laterally disposed. The roentgenogram (*b*) represents this final stage in the development of the pharyngoesophageal diverticulum. Note particularly the fact that the barium will first fill the sac and then spill over into the lateral slit which now represents the opening into the true esophagus. This roentgenogram demonstrates (1) the value of the oblique view in the study of these diverticula and (2) the dangers inherent in indiscriminate esophagoscopy or bouginage. (From: LAHEY, F. H. and WARREN, K. W. Esophageal diverticula. *Surg., Gynec. & Obst.*, 98: 1–28, 1954.)

of the diverticulum, the opening of the esophagus is converted into a lateral slit and the opening into the diverticulum comes to lie in the transverse plane. Swallowed food and liquid thus pass directly into the diverticulum rather than into the laterally placed opening of the true esophagus.

SURGICAL TREATMENT

The surgical treatment of pulsion diverticulum of the pharyngoesophagus has gone through many interesting phases. Early proposals included that of Bell of establishing an external fistula to empty the diverticulum. Kluge [4] in 1850, suggested excision of the diverticulum, which was first done in 1884 by Niehans. Girard [5], in 1896, advocated invagination of the diverticulum. Schmid [6] proposed diverticulopexy in 1912, the rationale being that if the tip of the sac were anchored at a point higher than the neck of the diverticulum, food would not accumulate within the sac.

The outstanding characteristics of the early historical treatment of this lesion were the high mortality and the high recurrence rate. The mortality was particularly high in the one-stage procedure. As a consequence, Goldmann [7], in 1909, performed the first two-stage operation. This consisted of mobilizing the sac and ligating the pedicle with silk. The wound was then packed and the sac was fixed to the surface of the wound. In this instance the sac sloughed and on the eighth day a fistula formed which healed at the end of two months. In 1916

Murphy [8] modified this procedure by implanting the mobilized diverticulum into the wound without ligating the sac. Two weeks later the sac was resected. Lahey [9] reported a modification of the two-stage procedure in 1933. In those instances in which the sac was small, the diverticulum after mobilization was sutured to the sternohyoid muscle at a level superior to the neck of the diverticulum. When the diverticulum was large, the sac was implanted in the wound, again with the diverticulum at a level higher than its entrance into the pharyngoesophageal junction.

The two-stage procedure has been employed extensively at the clinic and its validity in terms of achieving highly satisfactory results as measured by mortality, morbidity, fistula formation and rate of recurrence justified its use. In a series of 365 pulsion pharyngoesophageal diverticula reported by Lahey and Warren from this clinic in 1954, there were but two deaths. The first patient, who was in the first series of twenty cases, was an eighty-four year old man who died of uremia four days after the first stage operation. In the second patient, a forty-eight year old man who was operated on more than ten years prior to the report, death was due to mediastinitis and empyema. In the same series of 365 cases there were only twelve recurrences.

COMPLICATIONS

The serious morbidity following operations for pulsion diverticula embraces fascial plane infection, wound abscess, mediastinal infection, persistent sinus tract, esophageal fistula, stricture of the esophagus and, more rarely, empyema or lung abscess. Leakage has occurred in only a very small number of patients in this series following the second stage removal of the sac, and in all but two patients showing such leakage, closure was effected spontaneously. In one patient a fistula occurred following a second stage removal. Gastrostomy was performed which resulted in spontaneous closure of the fistula in a short time. There has been no instance of empyema, deep cervical abscess or mediastinal infection (except the one fatal case mentioned previously). In one instance a stricture of the esophagus occurred which necessitated a secondary plastic enlargement of the lumen. This was accomplished successfully.

Despite these excellent results with the two-stage procedure there is an increasing tendency, as judged by reports in the literature, to elect a one-stage excision of pulsion diverticula of the pharyngoesophagus. With the relentless progress that is being made in every phase of surgical endeavor, with the attendant improvement in anesthesiology, the protection that is derived from antibiotic compounds and the more adequate preoperative and postoperative control of nutritional and electrolyte balance, it is no surprise that the one-stage procedure is growing in popularity. Harrington [10] and DeBakey et al. [11] have advocated the one-stage operation in the treatment of pharyngoesophageal diverticula. Recently, Sweet [12] reported on the one-stage surgical treatment of sixty-seven patients with pulsion diverticulum of the pharyngoesophagus. The advocates of the one-stage procedure emphasize the simplicity of the technic, the relative freedom from complications, the shorter hospital stay and the good long term results. In the series reported by Sweet there was only one recurrence and one esophageal fistula which closed spontaneously at the end of three months.

It is, of course, difficult to compare series of cases which are not basically comparable in the very important respect of total numbers. If an attempt is made to compare a segment of the experience accumulated at the clinic, arbitrarily selected to correspond more precisely with the chronologic period in which the majority of the advocates of the one-stage procedure have recorded their results, it could be pointed out that in the clinic series there have been no postoperative deaths in the last 300 cases.

Realizing, as we do, that the operation of removal of a pulsion diverticulum of the pharyngoesophagus can at one time be a relatively simple procedure when the sac is small, easily identifiable, readily mobilized, with a small and distinct neck, and recognizing at other times that dissection of these large diverticula with marked peridiverticulitis and with adherence of the sac to the mediastinal or parietal pleura or to the longitudinal segment of the esophagus below the neck of the sac can be extremely demanding, it is obvious that in many instances a one-stage procedure can be done safely and effectively and without undue risk to the patient, particularly by a surgeon experienced in the technical manipulation of the esophagus. However, there remains with

us the continuing conviction that the two-stage procedure has considerable merit in many instances and that this operation should not be summarily discarded. It should be stated, however, that at present a large majority of esophageal diverticula operated upon at the clinic are removed in one stage.

TECHNIC

In this communication the technic of both the one- and the two-stage excision of the diverticulum will be described.

The Incision. While it is possible to remove a pharyngoesophageal diverticulum through one of three classic cervical incisions, namely, (1) a modification of the usual goiter incision with the left angle of the incision turned superiorly along the anterior border of the sternomastoid muscle, (2) a transverse incision in line with the skin cleavage placed over the approximate position of the neck of the diverticulum, or (3) the long longitudinal incision anterior and parallel to the anterior edge of the sternomastoid muscle, we have employed the long longitudinal incision routinely at the clinic. Since these diverticula, as previously stated, vary so widely in terms of technical difficulties involved in exposing, delivering and dissecting the neck of the sac, we do not compromise with the length of the incision. It is extremely important in patients with large diverticula in which the tip of the sac has extended into the mediastinum to assure at the outset that the length of the incision will be adequate to meet all technical demands that may be encountered. Lahey recorded that on more than one occasion he had removed a segment of adherent pleura in mobilizing the lower portion of the sac as it was dissected out of the mediastinum. The apex of the lung itself may be attached to the tip of the sac as a result of extensive inflammation and peridiverticulitis. When one has encountered some of these unusual complications in the management of this lesion, one learns to appreciate the value of wide anatomic exposure which is afforded by a long longitudinal incision. The location and extent of the incision as well as the scope of exposure are demonstrated in Figure 4. After the sternomastoid muscle has been retracted laterally, the anterior belly of the omohyoid comes into view. This muscle is divided between clamps and its anterior portion removed.

Removal of the anterior belly of the omohyoid gives immediate access to the internal jugular vein and exposes the middle thyroid veins. These veins are divided between clamps and ligated. Following the division of these tributaries of the internal jugular vein, the edge of the thyroid can be elevated out of its bed by blunt retractors. With the common carotid artery and the internal jugular vein pulled laterally and the thyroid lobe elevated anteriorly and medially, the inferior thyroid artery can be demonstrated, as can the recurrent laryngeal nerve which crosses the artery at right angles, usually deep to the artery, approximately in the groove between the trachea and the anterior wall of the esophagus. It is wise to visualize the upper portion of the recurrent nerve. The sac of the diverticulum is deep to the inferior thyroid artery. This artery, therefore, is divided between clamps and doubly ligated. The diverticulum can then be visualized. By gentle traction upon the diverticulum with the aid of Babcock forceps, the junction between the inner edge of the sac and the longitudinal esophagus can be demonstrated. The posterior aspect of the sac can be elevated from the prevertebral fascia.

When the sac has extended into the mediastinum, it must be dissected from the longitudinal esophagus with great care. This dissection will be facilitated by picking up the readily available portion of the sac with Babcock forceps; by careful dissection and traction the lowermost part of the diverticulum can be gradually elevated. Replacing the Babcock forceps lower and lower as the dissection proceeds is an extremely helpful maneuver in this regard.

In the early experience in the treatment of these diverticula the extent of the envelopment of the sac by fibers of the cricopharyngei may lead to considerable confusion. This investment will be meager in some sacs while in others considerable dissection may be necessary to separate the diverticulum from the longitudinal esophagus because of the density of the cricopharyngeal fibers enveloping it. The sac in its descent along the longitudinal esophagus or into the mediastinum carries with it a complete covering or investment of muscular fibers of the cricopharyngei, and even after the sac has been completely liberated so that it appears to be thoroughly mobilized, it will still be necessary to dissect circumferentially at the neck the cricopharyngeal fibers

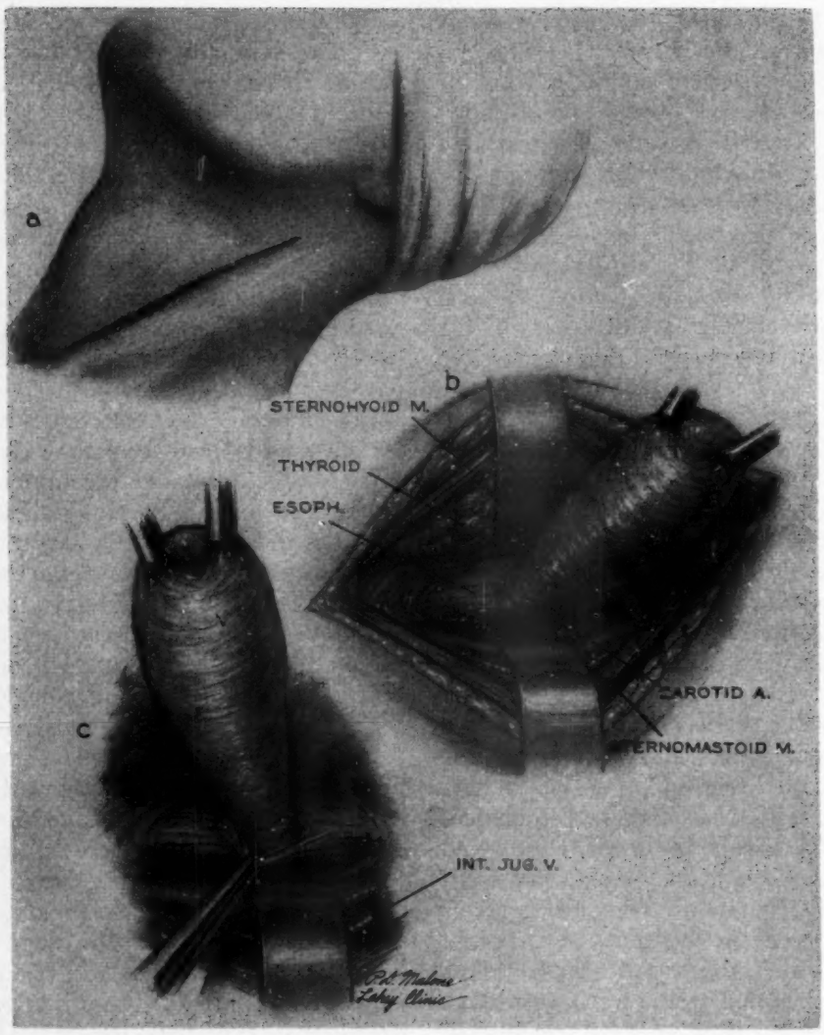

FIG. 4. (*a*) The long longitudinal incision in front of the sternomastoid. (*b*) The wide exposure obtained by retraction of the common carotid, internal jugular and sternomastoid; the thyroid gland is lifted out of the way by the second retractor. (*c*) A semi-diagrammatic drawing of how the neck of the sac can be exposed in this wide and long incision. This interpretation is a little misleading in that it suggests that the sac comes off the left side of the esophagus instead of the midline in the back, as mentioned in the text, but this distortion is necessary for the completeness of the exposure. (From: LAHEY, F. H. and WARREN, K. W. Esophageal diverticula. *Surg., Gynec. & Obst.*, 98: 1–28, 1954.)

until the pale white submucosa of the sac is clearly visible. This particular phase is perhaps the most dangerous aspect of the technic of excision of an esophageal diverticulum, especially if a two-stage procedure is elected. It is usually easy to divest the neck of the sac of the encircling fibers of the cricopharyngei on the superior and left lateral aspects, but it is more demanding and sometimes extremely hazardous to achieve with equal thoroughness complete dissection of the inferior and especially the right side of the neck of the diverticulum as it emerges from the longitudinal esophagus. The difficulty in visualizing and dissecting the right side of the neck of the sac is a reflection of the inability to rotate this segment of the esophagus in such a manner as to bring the right side of the esophagus well into view. Because of the difficulty or the impossibility in most instances of visualizing the right side of the neck of the sac, it is our practice, in performing the two-stage procedure, not to insist upon complete dissection of the right side at the first stage. Every effort is made, on the other hand, to

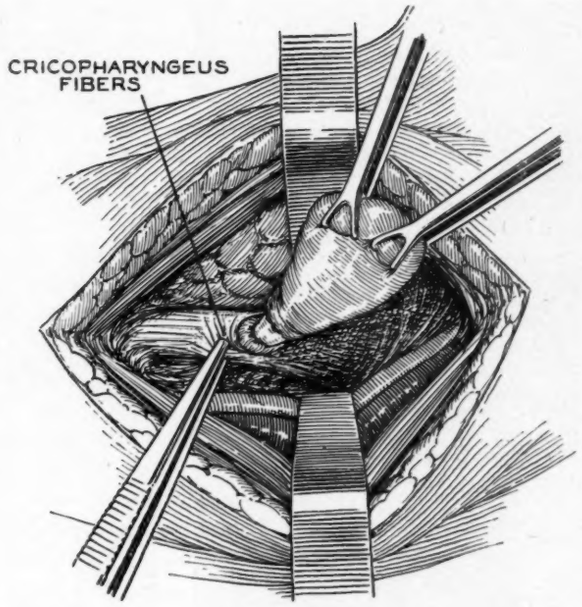

FIG. 5A. This semi-diagrammatic illustration shows the neck of the sac completely freed of muscle fibers and consisting solely of mucosa and submucosa. Note also the sling fibers demonstrated at the lower angle of the neck of the sac. (From: LAHEY, F. H. and WARREN, K. W. Epophageal diverticula. *Surg., Gynec. & Obst.*, 98: 1–28, 1954.)

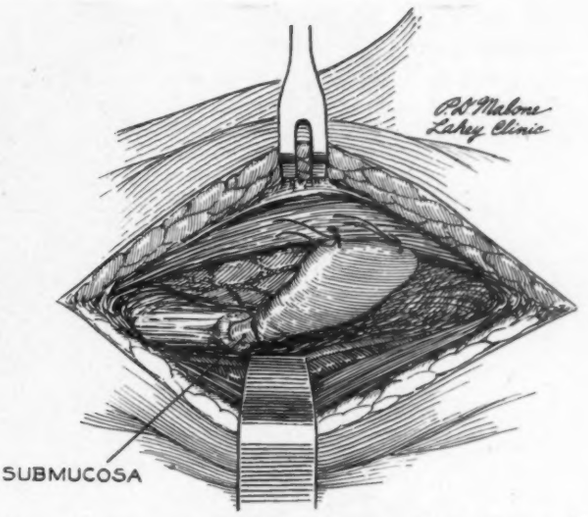

FIG. 5B. The neck of the sac is completely dissected above, in front and below, but the posterior layer of muscle is still unremoved, to be left to the second stage. Note also that this illustration, in which the tip of the sac is fixed to the edge of the sternohyoid between the first and second stage, gives the impression that the sutures pass through the wall of the sac. In reality they include only the adventitious tissue at the tip of the sac. Great care is exercised that the needle actually does not penetrate the wall of the sac because, as stated in the text, if it does leakage and soiling will be produced. (From: LAHEY, F. H. and WARREN, K. W. Esophageal diverticula. *Surg., Gynec. & Obst.*, 98: 1–28, 1954.)

delineate the submucosal layer at the upper and lower angles of the neck on the left side. The main objective of the first stage is to mobilize the sac completely, particularly at the inferior angle, so that the acute angle described by the sac of the diverticulum and the longitudinal esophagus in the preoperative state is converted into an obtuse angle, and thus there is no tendency for food to enter the sac during its suspension between stages. Not only is it safer to defer the final phase of the dissection on the blind side of the neck to the second stage when the fascial planes of the neck and mediastinum have been sealed, if the two-stage procedure is elected, but also it is actually easier at the second stage to wipe back the remaining fibers of the cricopharyngei on the right side of the neck and at the superior angle where frequently a troublesome plexus of veins will be encountered. (Fig. *5a.*) Perhaps it is, as some think, an inconsequential point to insist upon this thorough demonstration of the submucosa at the junction of the neck of the diverticulum with the esophagus in every instance, but it is our concept that the low incidence of recurrence reported in this and previous communications is due in large measure to this insistence.

An additional technical consideration of some importance which is frequently overlooked is the division of the cricopharyngei fibers directly beneath the lower angle of the neck of the sac. If these fibers are not divided it is unlikely that the true junction of the neck of the sac with the longitudinal esophagus will be observed, and also unlikely that the ligature of the sac at the second stage will be sufficiently close to this junction to prevent recurrence. Being aware of the importance of these sling fibers makes their division relatively simple. They may be elevated on a small curved clamp and divided down to the level of the submucosal layer.

Once the neck of the sac has been dissected in this manner, the adventitious tissue about the tip of the sac or the fibers of the cricopharyngei at this point are picked up with the small clamps, exercising care not to include the wall of the sac itself, and these tissues are ligated with fine silk. The tip of the sac is then

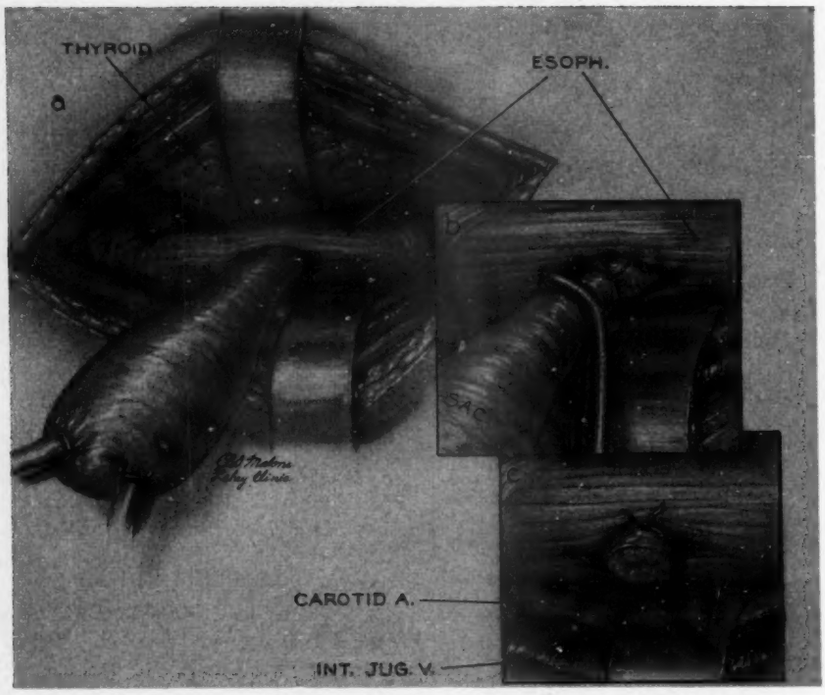

FIG. 6. (*a*) The exposure of the neck of the sac. The origin of the sac is in the midline directly behind and not as appears in this illustration coming off the left wall of the esophagus. This distortion is necessary in order to depict the actual exposure of the neck of the sac. (*b*) The same distortion with the neck of the sac to the left, the accurate ligature of the sac neck level with the esophagus itself. The right angle clamp is placed at a proper distance from the ligature to prevent soiling of the wound from the contents of the sac. (*c*) The neck of the sac is severed. Note that an adequate amount of sac neck still remains so there is no danger of the ligature slipping (see text). (From: LAHEY, F. H. and WARREN, K. W. Esophageal diverticula. *Surg., Gynec. & Obst.*, 98: 1–28, 1954.)

anchored to the outer edge of the sternohyoid muscle by transfixing these ligatures to this area of the muscle. Thus, the sac is suspended so that the tip of the sac is cephalad and the neck, at the junction with the longitudinal esophagus, is the most dependent portion of the diverticulum. These two black silk sutures are cut long enough to aid in identification at the second stage. (Fig. 5*b*.) When the sac is large it will be impossible to suspend the diverticulum in the manner described. Under such circumstances it will be necessary in the two-stage procedure to implant the sac in the wound itself. When this method is employed, great care should be taken to place the mobilized diverticulum sufficiently high in the longitudinal incision in the neck so that the tip of the sac is upward, to discourage the entrance of food into the diverticulum.

A cigarette drain is inserted into the fascial planes at the lower angle of the wound or into the mediastinum if the sac is large, and the platysma is approximated with fine catgut. The skin is closed with Michel's clips.

Second Stage. The second stage procedure is done seven days after the initial operation. This seems to be the most appropriate interval. It is a simple matter to separate the edges of the original incision by manipulation with the index finger or by employing the blunt edge of the handle of the scalpel. One should attempt to follow the line of separation through which the first stage was done, by retracting the common carotid and internal jugular vein laterally and the thyroid, trachea and esophagus medially. Once more the thyroid is elevated from the wound by the aid of a blunt retractor. With the carotid and internal jugular vein retracted laterally and the thyroid medially, the tip of the suspended sac comes into view at the outer edge of the sternohyoid muscle. The sac is mobilized by cutting the stitches,

and the neck of the sac is carefully delineated by gently wiping the fibers of the cricopharyngei away from the neck of the sac with a wad of gauze in a right-angle clamp. It is at this point that the remaining fibers of the cricopharyngeal muscle at the right aspect of the neck of the sac are carefully wiped away from the junction of the neck of the sac with the longitudinal esophagus. The entire circumference of the exposed neck of the sac should consist only of submucosa and mucosa. The neck of the sac is then ligated with No. 0 chromic catgut. The catgut ligature is placed as near the junction of the neck of the sac with the longitudinal esophagus as possible. This maneuver is aided by having the assistant exert gentle traction on the diverticulum while this ligature is being tied. Once this ligature is in place, a right-angle clamp is applied distal to the ligature and the sac is amputated.

A word of caution should be interjected at this point. One should avoid amputating the sac too close to the previously placed ligature. A generous rosette of mucosa and submucosa is best left distal to the ligature to prevent slipping. (Fig. 6.)

Following the amputation of the sac, a cigarette drain is inserted into the depth of the wound and brought out at the lower angle of the incision, and the incision is closed in layers. A Levin tube, which has been guided into the lower aspect of the esophagus and then into the stomach at the time of dissection of the neck of the sac, is left in place for one week for feeding purposes. The drain is removed on the fourth postoperative day, and the Levin tube is withdrawn on the seventh postoperative day. The patient is permitted to eat immediately following removal of the Levin tube and is discharged from the hospital the same day.

In many instances in which the diverticulum is large, the neck of the sac will be of such large caliber that it does not lend itself to the ligature technic, as described previously. Under these circumstances it is preferable to amputate the sac and to close the opening in the esophagus by a continuous, longitudinal inverting suture of fine chromic catgut reinforced by interrupted sutures of the same material. (Fig. 7.) In those unusual instances in which the caliber of the neck of the sac is extremely large in comparison to the circumference of the longitudinal esophagus, it is wise to amputate the sac and then to close the opening in the esophagus transversely. This, again, may be accomplished by a continuous inverting suture of fine chromic catgut, reinforced by interrupted sutures of the same material, or the opening can be closed with two layers of interrupted sutures.

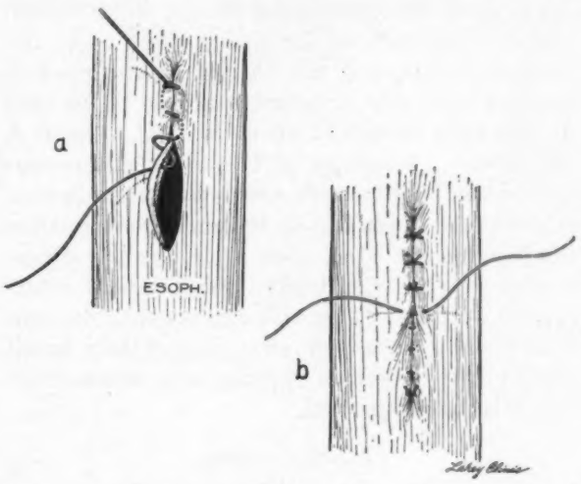

FIG. 7. (*a*) The opening into the esophagus is closed with a continuous inverting suture of fine chromic catgut and reinforced (*b*) with interrupted sutures of the same material.

One-stage Excision. The technic for a one-stage excision of a pharyngoesophageal diverticulum is, in most respects, similar to the two-stage procedure up to the point of management of the neck of the sac. The same generous longitudinal incision is used, and the exposure of the diverticulum and the neck is achieved in the same fashion, the essential difference being that, in the one-stage procedure, dissection of the cricopharyngei fibers at the junction of the neck of the sac with the longitudinal esophagus is completed in its entire circumference. The submucosa is exposed on the posterior or right side of the junction of the neck of the sac with the esophagus just as it is in the first stage at the superior, inferior and left side of the neck of the sac. Again it should be emphasized that the well developed fibers of the cricopharyngeal muscle at the inferior angle of the neck of the sac with the esophagus should be carefully dissected and that these fibers, in turn, should be elevated on the point of a fine curved clamp and should be carefully divided, thus eliminating the sling effect which we believe is such an important consideration in possible recurrence of the diverticulum. Once the circumferential

dissection has been accomplished down to the submucosa, the surgeon decides what method he will use to deal with the neck of the sac. He can choose either simple ligature as described previously, with amputation distal to the ligature, or removal of the sac and closure of the opening in the esophagus in the longitudinal plane if the neck of the sac is small or in the transverse plane if the caliber of the neck is large. The choice of suture material to be used in this area is largely an individual matter. A significant advantage of the one-stage operation is the facility with which the cricopharyngeal muscle fibers can be reapproximated, thus reinforcing the weak spot at which the diverticulum occurs. Certainly, two layers of interrupted sutures of fine silk are acceptable, and if fine silk is used on an appropriately small needle, the chance of causing any stricture at this level is minimized.

COMPLICATIONS

Injury to the recurrent laryngeal nerve occurred in twelve of approximately 405 cases of pharyngoesophageal diverticula treated at the clinic. Most of these injuries occurred in the early stages of experience with this operation and are now avoided by careful dissection of the recurrent laryngeal nerve.

There has been no instance of injury to the thoracic duct in this series, even in the large number of patients with mediastinal extension of the diverticulum. In one patient referred to the clinic, an esophagoscope had been passed through the tip of the sac. Following the injury a gauze pack had been inserted into the neck through a long longitudinal incision, and the patient then referred to the clinic. When an attempt was made to remove the pack, massive hemorrhage occurred into the wound. The pack was reinserted, with control of the hemorrhage, and the patient taken to the operating room. When the pack was removed, it was observed that the massive bleeding came from an opening in the wall of the internal jugular vein at a point well below the clavicle. Lahey has described the method of management of this particular hemorrhage by dissecting the jugular vein from the carotid artery in the mid-neck and dividing the vein between clamps. The upper end of the jugular vein was ligated and the lower end was clamped and twisted until the torsion of the vein passed the opening in the wall, thus controlling the bleeding. A small right-angle clamp could then be placed upon the twisted vein below the level of the opening in its wall, and the vein ligated.

There have been thirteen recurrent diverticula in 405 patients operated on at the clinic. A small number of patients had some leakage following the second stage removal of the sac, but in all but two cases closure occurred spontaneously without further surgery and in only one of the two was surgical intervention necessary to obtain closure. There have been no persisting sinuses.

ONE-STAGE VERSUS TWO-STAGE EXCISION

It is increasingly apparent that one-stage excision of esophageal diverticula is becoming more and more popular. This popularity will undoubtedly grow as larger and larger series are reported showing mortality, morbidity and recurrent rates approaching comparable rates for the two-stage procedure described in this article and in the article by Lahey and Warren in 1954 [*1*]. With the dramatic progress that is being made in all fields of surgery, it is inevitable that results equal or superior to this series in these several respects shall be recorded. These initial achievements by the one-stage procedure will be accomplished and recorded primarily by that distinguished group of surgeons who have had a wide experience in esophageal surgery. For the surgeon with limited experience in this field, the validity of the two-stage procedure should still have meaning. Currently, 90 per cent of the esophageal diverticula removed at the Lahey Clinic are done in one stage.

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Diverticula of the Thoracic Esophagus

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DURING the past decade experience with diverticula of the thoracic esophagus has shown that these lesions (Fig. 1), particularly the epiphrenic type, not infrequently present serious problems in diagnosis and management. In a significant number of cases clinical symptoms are often due primarily to associated esophageal lesions such as hiatus hernia, cardiospasm or diffuse esophageal spasm; the symptoms often cannot be differentiated from those due to the diverticulum [1–3].

Diverticula of the thoracic esophagus are usually classified according to their anatomic position as epibronchial and epiphrenic. The epibronchial diverticula are usually of the traction type and the epiphrenic diverticula are almost always of the pulsion variety. The former lesions seldom produce symptoms and are rarely of clinical importance unless there is secondary abscess or fistula formation. The epiphrenic diverticula are the most important; an untreated diverticulum in this region may seriously jeopardize the patient's health and life, and non-operative measures may offer the patient, at best, slight temporary improvement.

The purposes of this paper are to present pertinent data relative to epibronchial traction diverticula and to discuss briefly the frequency, etiology and pathogenesis, clinical manifestations and treatment of epiphrenic diverticula. In addition, four illustrative cases with comments are presented.

EPIBRONCHIAL DIVERTICULUM
TRACTION TYPE

The traction type of diverticulum may be found in any part of the esophagus but occurs most commonly in the mid-esophagus at the level of the primary bronchi. These lesions are rather common. Starck [4] states that they are found in 5 to 6 per cent of all autopsies, most of which are of no clinical significance. They are true diverticula in that their walls contain all layers of the esophagus and consist of mucosa, submucosa and muscularis. Traction diverticula are believed to be caused by adhesions that arise from inflammatory processes in the adjacent lymph nodes. As a result, there is scarring and contraction of the inflamed tissue and the wall of the esophagus is dragged out in a tent-like fashion (Fig. 1A); consequently, a potential weakness develops in the esophageal wall. This weakness, coupled with the expanding intraluminal pressure, predisposes to diverticular formation.

These lesions usually originate in the anterior and lateral walls of the esophagus. They are frequently small, rarely exceeding 2 cm. in size. The fact that they are usually retracted laterally most likely explains why food seldom tends to collect in them. In addition, the presence of an intact muscular wall and a broad diverticular neck tends further to prevent food from accumulating and stagnating in its cavity.

In almost all of these cases there is an absence of symptoms. In the remaining cases the symptoms are usually dependent on the length of time that these diverticula retain food and the degree of infection that may exist. Under these circumstances, the symptoms are mild and consist mainly of substernal pain and burning. Dysphagia may constitute a prominent symptom. In rare instances, gross hemorrhage may result.

Diverticulitis is the most likely complication. In a microscopic study of forty cases of traction diverticula, Ribbert [5] found a high incidence of connective tissue replacement, increased intramural vascularity and mucosal thinning. Abscess formation with rupture into the mediastinum has occurred; in these cases the peripheral reaction which has been going on for some time tends to keep the process localized, and the possibility of a sudden free perforation

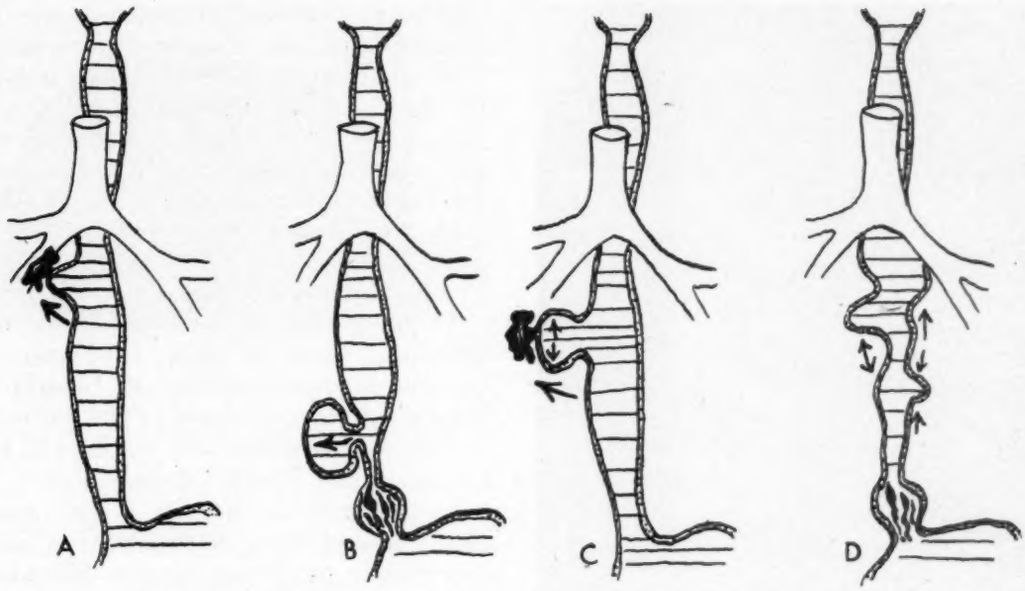

FIG. 1. A, traction diverticulum. Usually caused by inflammation of lymph nodes at bifurcation of the trachea, with ultimate adhesions to the esophageal wall. B, pulsion diverticulum. Usually caused by pressure within the esophagus most frequently found in the epiphrenic region. Hiatus hernia, diffuse esophageal spasm or cardiospasm are not infrequent associated lesions. Note the hiatus hernia and the postdiverticular stenosis. C, traction-pulsion diverticulum. This is a combination of the two generally encountered types (A and B). It is presumed that traction from the outer esophageal wall initiates a weakness and pulsion from intraluminal pressures causes a bulging of the wall. D, functional diverticulum. There is no unanimity of thought as to whether the changes in the esophageal wall are caused by local spasms (abnormal peristalsis) or traction upon the esophageal wall. (See Figure 2.)

of the diverticulum into the mediastinum is practically nil [6].

Occasionally, these lesions may lead to a fistulous communication. The exact location of the opening of the fistula into the respiratory tree was recorded in twelve of the fourteen reported cases of this type [7]. In eight of these there was a communication between the esophagus and a secondary bronchus (usually in the lower lobe). In four cases it communicated with the stem bronchus. The presence of a fistula which communicates with the lung may lead to the development of lung abscess or bronchiectasis. Abscess and fistula formation occur so rarely that it is necessary only to mention them.

The diagnosis of epibronchial traction diverticula is established by means of x-ray examination with the aid of barium swallow. Ordinarily, traction diverticula do not fill well with contrast media and it may be necessary to examine these patients in the recumbent or Trendelenburg position. If the presence of an esophagorespiratory fistula is suspected, lipiodol rather than barium should be ingested; iodized oil is preferable because it is less irritating to the tracheobronchial tree. Esophagoscopy and bronchoscopy may be necessary to rule out carcinoma, since malignant lesions account for about 50 per cent of all acquired communications.

The differential diagnosis of epibronchial traction diverticula includes pseudodiverticula, which are frequently associated with carcinoma, and the so-called transient or functional diverticula which arise from abnormal esophageal peristalsis. (Figs. 1D and 2.)

Since these lesions usually produce no symptoms, it is obvious that no active treatment is required in the majority of cases. If symptoms are present and there is esophagoscopic evidence of disease in the diverticulum, excision is indicated (Case 1). On rare occasions the presence of hemorrhage or esophageal obstruction secondary to diverticulitis may indicate surgery. Abscess or fistula formation with or without a communication between the diverticulum and lung may require, respectively, drainage of the mediastinum, repair of the fistula and segmental resection or lobectomy.

FIG. 2. Patient N. S. was admitted to the hospital because of dysphagia and substernal distress. X-ray reveals multiple diverticula and a hiatus hernia. The use of esophageal dilatations resulted in marked improvement. According to Johnstone [20], these multiple pouches are transient and disappear as the activity of the esophagus ceases; in the course of time some become permanent and are indistinguishable from traction diverticula. He states, "the behavior and the appearance of the esophagus, however, leaves no doubt as to the underlying lesion and the demonstration of a hiatus hernia supports the hypothesis of muscular incoordination."

EPIPHRENIC PULSION DIVERTICULUM

Epiphrenic diverticula occur most frequently in the lower esophageal region just above the diaphragm. (Fig. 1B.) These diverticula are usually referred to as the pulsion or pressure variety. They are considered true diverticula in that they contain all of the esophageal layers. These diverticula possess a muscular layer which is generally much attenuated; the wall consists mainly of mucosa, submucosa, muscularis mucosae and an outer fibrous layer [1]. This is unlike the traction type which possesses a well developed outer muscular coat.

Frequency. An approximate incidence of this condition is difficult to arrive at since a significant number of patients are asymptomatic and the diverticulum undiagnosed. Also, many are not reported; this is borne out by the fact that during the past three years three cases at Harlem Hospital and two cases at Columbus Hospital were treated and have not been reported in the literature. However, with the present day emphasis on routine x-ray examinations, this condition is likely to be recognized more frequently.

In 1933, Granet reported an incidence of forty-nine cases. Of these, thirty-one were of the true epiphrenic variety [8]. In 1934, Vinson collected forty-two cases at the Mayo Clinic, twenty-four of which were epiphrenic. In 1952, Goodman and Parnes [2] were able to collect from the literature a total of 126 cases; they also reported three of their own which were successfully treated by surgery. More recently, Habein, Moersch and Kirklin [1] were able to collect from the Mayo Clinic records, covering a ten-year period (1944–1953 inclusive), 124 cases of pulsion diverticula in the lower esophageal region.

Etiology and Associated Lesions. The etiology of epiphrenic pulsion diverticula is obscure. Most authors support view the that these lesions are congenital in origin. Carmen (quoted by Granet [8]) is of the opinion that in addition to the congenital basis, other factors must be included to explain their development and enlargement. They are, namely, the negative intrathoracic pressure, the positive intraesophageal pressure and the existence of a more complex anatomy in the lower esophageal region.

Other authors [10–14] are of the opinion that the presence of degenerative changes of the ganglia in the wall of the esophagus or lesions which cause esophageal obstruction, combined with the expanding pressure of swallowed foods, play a major role in their formation.

Raven [13] states, "the most reasonable supposition is the presence of distal esophageal obstruction with increased pressure, causing herniation of the mucosa in an area where muscle coats have been weakened by local esophagitis." Lahey and Warren [15] state, "this lesion, in all probability, however, is the result of a weakness in the lower end of the esophagus which must be largely limited to this epiphrenic point because no lesions are reported in the literature of this type above this level at the lower end of the esophagus." They

FIG. 3. Case II. Pulsion diverticulum consisting of thickened mucous membrane (a) of lower esophageal region. Note inflammatory reaction (b) and ulceration (c).

believe that this lesion is brought about in somewhat the same way as are pharyngo-esophageal diverticula by cardiospasm or fibrosis and the influence of increased intra-esophageal pressure.

McNealey and McCallister [*16*] report that these diverticula often are found proximal to a point of anatomic narrowing or functional narrowing (cardiospasm). In Vinson's [*9*] series of forty-two cases associated spasm occurred in eight; other associated conditions were diaphragmatic hernia, malignancy of the stomach, pyloric obstruction and duodenal ulcer. In Harrington's [*3*] series of eight cases treated by surgery, six had an associated esophageal hiatal hernia. Goodman and Parnes [*2*], in collecting 126 cases from the literature, conclude that about 65 per cent of the cases of epiphrenic diverticulum have an associated cardiospasm. Some authors [*17–19*] doubt this relationship since they have rarely encountered diverticula in patients with cardiospasm or achalasia. Johnstone [*20*] has examined more than 200 patients with achalasia without once finding an esophageal diverticulum. He also has examined a far greater number of patients with obstruction of the lower esophagus due to carcinoma, peptic ulcer, esophagitis and stricture and here, too, was able to find a true diverticulum only once. It has been postulated that cardiospasm may be secondary to the diverticulum, the result of reflex action originating in its inflamed wall [*2,18*].

These diverticula may be complicated by the presence of inflammation (diverticulitis) or ulceration. (Fig. 3.) Occasionally, there is an associated esophagitis. On rare occasions perforation, hemorrhage and even malignancy may be encountered.

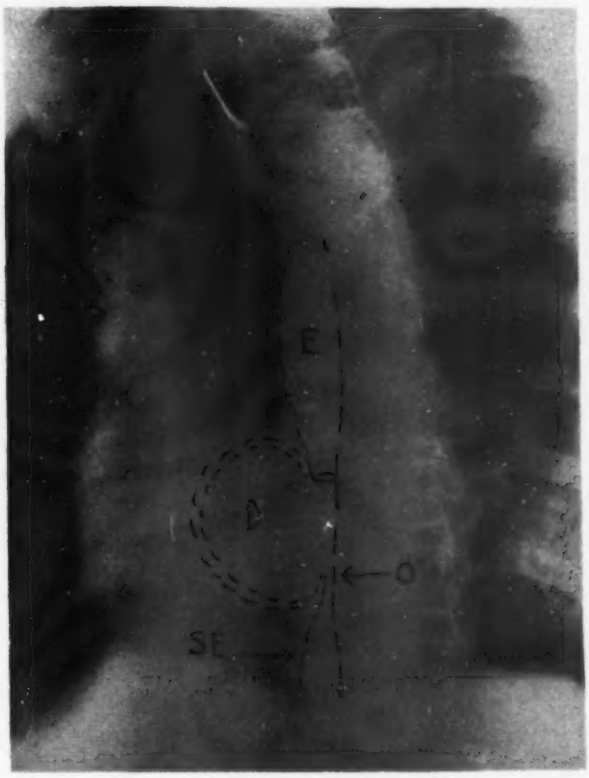

FIG. 4. In advanced cases of epiphrenic diverticula, almost a complete mechanical obstruction of the esophagus occurs (E). If the sac (D) is not removed, the patient will require esophagoscopic dilatations (O) to maintain an adequate passage. Note the adequacy of the luminal diameter of the subdiverticular esophagus (SE).

Clinical Picture. The symptoms of epiphrenic pulsion diverticula are most variable. In a significant number of cases there is an absence of symptoms. In the majority of cases the severity varies from mild dyspepsia to marked dysphagia or regurgitation. The regurgitation of accumulated food frequently occurs when the patient assumes the recumbent position. Discomfort and pain are infrequent manifestations; they are usually referred to the substernal or epigastric regions. In the more advanced stages, when almost a complete mechanical obstruction of the esophagus exists, weakness, loss of weight and malnutrition are encountered (Case III). (Fig. 4.) Respiratory tract symptoms may be present; on occasion, clinical manifestations of cardiac disease may occur.

The diagnosis is made by x-ray examination, with the aid of a barium swallow. The lesion is

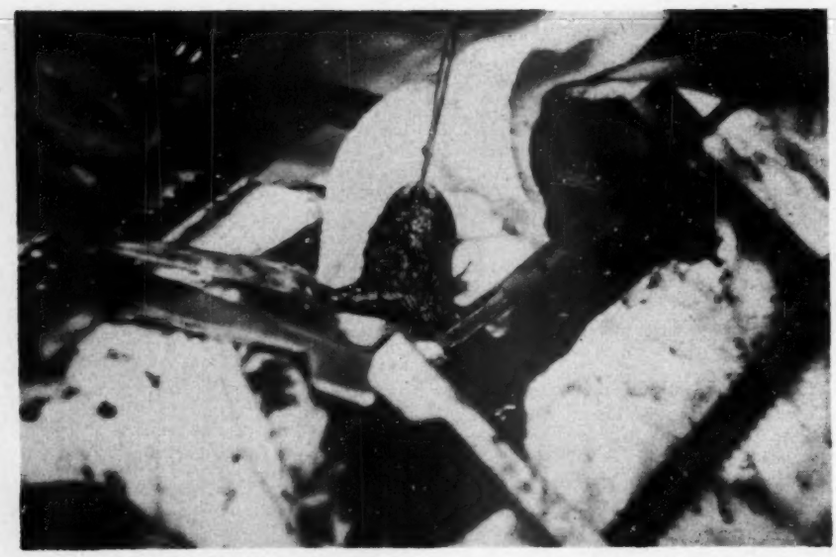

FIG. 5A. The epiphrenic diverticulum can be removed either through a right or left standard thoracotomy incision with removal of the seventh or eighth rib. The mediastinal pleura is separated sufficiently from the esophagus to permit adequate exposure of the diverticulum. Two Penrose drains are used to steady the diverticulum. The success of the operation is dependent upon the avoidance of trauma and the utilization of fine suture material.

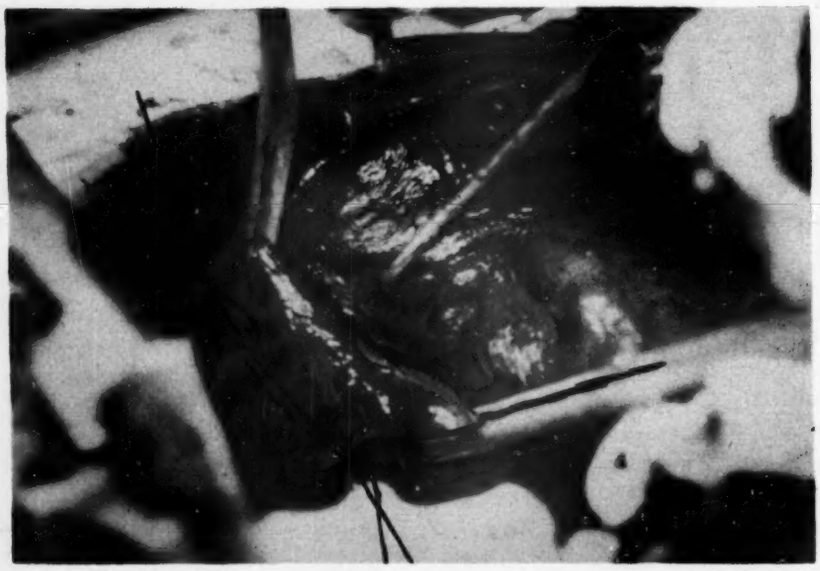

FIG. 5B. After the neck of the sac has been completely separated, the outer layers of the diverticulum are cut. An important detail in the treatment of esophageal diverticula is to make allowance for an adequate closure without jeopardizing the luminal diameter of the esophagus. The incision is made at an appreciable distance from the base of the sac. Stay sutures are placed at both ends. The longitudinal defect is then closed in two layers with fine suture material. The mucosal layer can be approximated with either catgut or silk; the muscular layer is brought together with fine silk. A third layer may be inserted. In some instances, a flap of mediastinal pleura may be sutured over the closure.

nearly always seen on a routine, posteroanterior chest film. Occasionally, the diverticula may not be easily visualized due to the presence of food or liquid in the diverticulum or because of an occlusion of the neck of the diverticulum, which results from associated esophagitis and spasm. Esophagoscopy may be necessary to rule out carcinoma. This procedure proves helpful in determining the presence of esophagitis and other possibly associated lesions, e.g., spasm, stricture and hiatus hernia.

Treatment. In patients with asymptomatic lesions no therapy is necessary. If symptoms should develop, lavage and dietary management are indicated. The sac can usually be washed out with a glassful of water after each meal. The diet should consist of soft, bland foods. If ulceration exists, antacids are indicated. Symptomatic relief can be expected in patients with spasm of the esophagus by the use of esophageal dilatations. If these measures fail, operation is indicated. In most instances surgery proves to be the only effective form of treatment. However, in the group of patients with an associated lesion, e.g., esophageal hiatus hernia, diffuse spasm or cardiospasm, the results following diverticulectomy may be uniformly poor unless the associated lesion is also treated (Case IV). In a recent review it was emphasized that the presence of an associated lesion should weigh against surgical intervention for treating the diverticulum, unless the associated lesion can be managed surgically at the same time or prior to diverticulectomy by either medical or surgical means [21].

Numerous surgical procedures have been performed for the treatment of epiphrenic diverticula [2,22], namely, gastrostomy, diverticulogastrostomy, invagination, extrapleural resection of the sac, diverticulopexy and transthoracic resection of sac and direct suture of the esophageal wall. Of these, the present accepted method is the transthoracic approach with excision of the diverticulum (Figs. 5A and B.)

On occasion, in elderly, malnourished, poor risk patients, diverticulopexy may be justified. This procedure produces satisfactory results [23]. The other procedures are enumerated only to mention the earlier indirect methods of attack. More recently, esophagogastrostomy has been utilized for the surgical treatment of epiphrenic diverticulum and an associated lesion. In one case, there was an associated

FIG. 6. Case 1. At operation the diverticulum was found to be of the traction-pulsion variety.

esophageal atresia [2]. In the other, there was a stricture formation which extended beyond the neck of the diverticulum to involve the segment of esophagus just distal to the pouch [22].

CASE REPORTS

CASE 1. G. G., a forty-five year old white man, was admitted to Columbus Hospital on January 5, 1951, in moderate distress from chest pain, nausea and hiccoughing. He also complained of headaches and general lassitude. The remaining complaints were bizarre and were suggestive of a psychoneurotic state.

A complete work-up, urinalysis, complete blood count, blood chemistry studies and electrocardiographic tracing revealed no abnormalities. Gastrointestinal series revealed an esophageal diverticulum in the region of the bifurcation of the trachea. (Fig. 6.) At esophagoscopy, the opening of the diverticulum was visualized at approximately 28 cm. distal to the central incisor teeth; its neck measured about 2 cm. in diameter. The esophagoscope was easily introduced into the diverticulum;

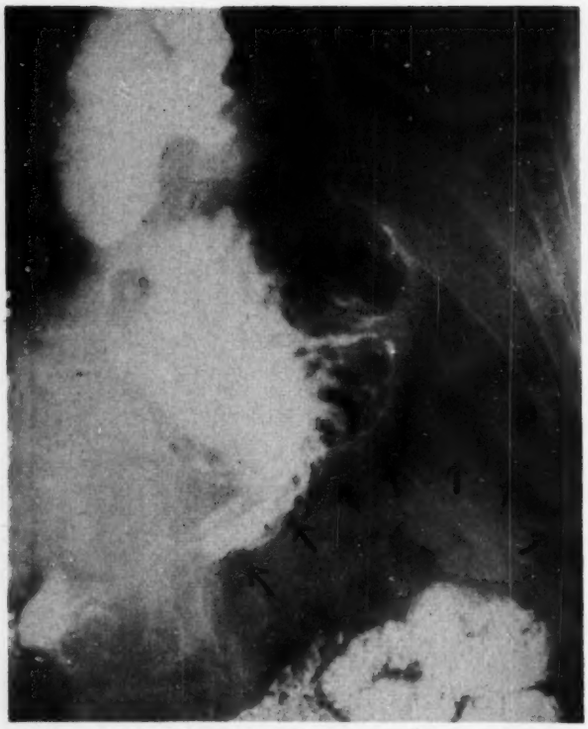

FIG. 7. Case II. Esophagram taken on August 16, 1956, shows a large pulsion diverticulum in the lower part of the esophagus and a diaphragmatic hernia, hiatus type. X-rays taken five years previously showed the same lesions; they were smaller in size. There were never any symptoms.

there was evidence of inflammation of the mucous membrane. In the most dependent area, the lining bled easily when manipulated with a sponge which was attached to a sponge carrier.

On January 21, 1951, through a right transpleural incision, the posterior mediastinal pleura was incised in the region of the diverticulum. The esophagus and the protruding diverticulum were readily visualized and easily dissected free from the adjacent organs. It was held by a few adhesions. The sac arose from the posterolateral aspect of the esophagus and passed to the right. Its fundus was in a dependent relation to the esophagus and the innermost mucosal layer protruded through the smooth, oval defect in the esophageal muscular coats. The diverticulum was excised. The operative technic is illustrated in Figures 4A and B.

The patient's postoperative course was uneventful. He was discharged on the eleventh day. Six weeks later he felt much improved. A recent examination of the patient, five years after surgery, revealed that he was in excellent physical condition and no longer had any of the symptoms which were present prior to diverticulectomy. Barium swallow revealed no residual defect in the region where the diverticulum was excised.

Comment: This case presented a difficult problem in diagnosis and management. At the time of the patient's admission to the hospital, he was labeled a psychoneurotic. However, in view of the history, esophagoscopic findings and the fundal dependency of the diverticulum, it was decided that the patient should be subjected to surgery despite the fact that the lesion was epibronchial in position and most likely of the traction type. At surgery, a traction-pulsion diverticulum was found. (Fig. 1C.)

The patient rallied most satisfactorily and today is in an excellent state of health. This case demonstrated that many of the symptoms were directly attributable to the presence of the diverticulum, and while of little clinical importance, surgery proves helpful in disclosing the type of diverticulum that is present.

CASE II. M. K., a sixty-two year old white man, was admitted to Columbus Hospital on August 16, 1956, because of a "general run-down feeling." The past history was essentially negative except that five years prior to admission the patient experienced a sudden attack of substernal pain which radiated to the precordial region and lasted for about two and a half hours. At that time a complete work-up, urinalysis, blood studies including chemistry, and electrocardiographic tracings were essentially negative. A barium swallow and gastrointestinal series, however, revealed the presence of an epiphrenic pulsion diverticulum of the esophagus and a diaphragmatic hiatus hernia.

The recent work-up revealed no evidence of abnormalities except the presence of the pulsion diverticulum and a diaphragmatic hiatus hernia. (Fig. 7.) These lesions were both present five years previously; both have increased appreciably in size. After three days in the hospital the patient was discharged, at which time he stated that he felt much improved.

Comment: This case clearly demonstrates that a pulsion diverticulum, even though it becomes progressively larger over a period of time, may do so without causing any significant symptoms. In approximately one-third of these

cases, there are no symptoms. The incidence of the coexistence of a diaphragmatic hiatus hernia and a pulsion diverticulum in the epiphrenic region is relatively high. In one reported case a diaphragmatic hiatus hernia, associated with diverticulum, so confused the picture that the presence of a diverticulum was not confirmed until after the hernia had been repaired abdominally [*1*].

CASE III. E. W., a forty-nine year old Negro woman, was admitted to Harlem Hospital on December 29, 1954. Her chief complaint was vomiting immediately after eating a large meal. On occasion, she vomited even small amounts of food and felt a knot in the epigastric region. There was a weight loss of 20 pounds. On the day prior to admission she was in acute distress, complaining of unbearable discomfort in the upper abdomen. The work-up was significant in that she was anemic and had left ventricular damage as evidenced by electrocardiographic studies.

On December 30, 1954, at 10 A.M., the esophagram demonstrated a large diverticulum of the lower third of the esophagus, about 5 cm. above the diaphragm. (Fig. 8.) It pointed to the right and slightly posterior. There was no evidence of a hiatus hernia.

At 7 P.M. that evening, because of marked substernal distress, difficulty in breathing and continual regurgitation of thin barium and secretions, a large bore tube was passed into the esophagus; 300 cc. of barium and fluid were aspirated. The patient was immediately much relieved. The procedure was repeated on January 1st and January 7th.

On January 10, 1955, esophagram with fluoroscopic examination showed the previously reported diverticulum in the lower third of the esophagus. With the patient in the Trendelenburg position, the contents of the diverticulum coursed back into the upper esophagus. A small collection of barium was also present at the cardia, which was suggestive of a hiatus hernia. The examination was made in the direct posteroanterior position, with the patient in marked Trendelenburg position.

On January 12, 1955, esophagoscopy was performed. The opening of the diverticulum was encountered at approximately 32 cm. from the central incisor teeth. The pouch deviated to the right; its neck measured about 1 cm. in diameter. It was difficult to remove all the debris and barium from the diverticulum by endoscopic aspiration. For this reason the patient was subjected to two additional endoscopic aspirations.

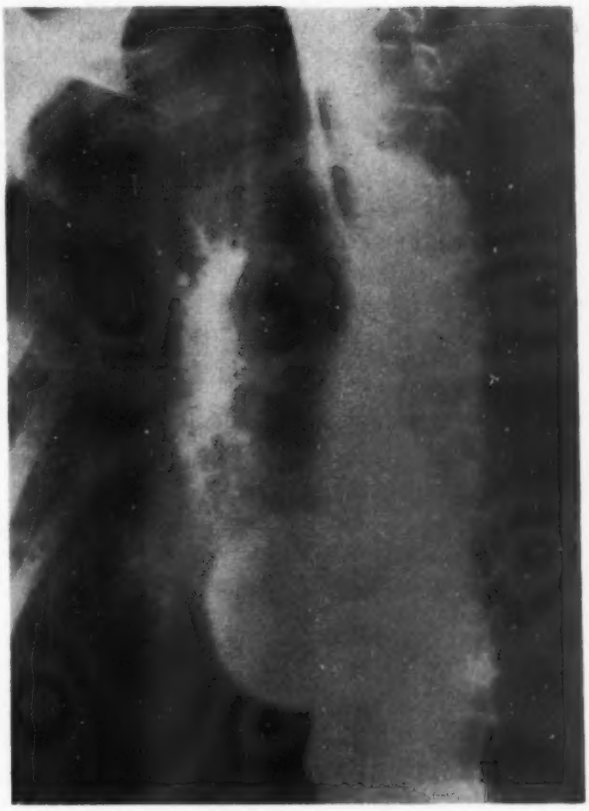

FIG. 8. Case III. Preoperative esophagram taken on December 30, 1954. Patient underwent excision of the diverticulum for obstructive symptoms. Postoperative esophagram taken on February 15, 1955, revealed no evidence of diverticulum, and there was no obstruction to the passage of barium through the esophagus.

She was given intravenous fluids, vitamins and two blood transfusions in preparation for surgery.

On January 15, 1955, the patient was placed in the right lateral position and the chest was entered through the bed of the eighth rib. The diverticulum was found protruding from the anterior lateral wall of the esophagus. It was directed to the right and was 8 cm. above the cardia in the retrocardiac area. The wall of the sac was adherent to the surrounding structures and pressed against the lower esophagus and the right pleura. The sac was dissected free by blunt and sharp dissection. When the sac was completely freed, it averaged about 8 cm. in diameter and the neck was fairly broad. (Fig. 4A.) The diverticulum was then cut at its base, leaving enough esophagus so as to avoid

the danger of constricting the lumen. (Fig. 4B.) The longitudinal opening was then closed with fine catgut for the inner layer and fine silk for the outer layer. Postoperative management was essentially that which is routinely instituted for any transthoracic procedure.

Postoperatively, the patient recovered rapidly. Parenteral fluids containing glucose, saline and vitamins were given for the first three days, with occasional sips of small amounts of water. On the fourth postoperative day, the patient was given a liquid diet. Soft, solid foods were begun on the seventh day. The patient was discharged from the hospital on the sixteenth postoperative day, at which time she was able to swallow soft foods without any difficulty. The patient was again examined a year later. She stated that she no longer had any of the symptoms which were most annoying and incapacitating prior to surgery.

Comment: This case constitutes the classic indication for surgery. Excision of the diverticulum was the operation of choice. There was an associated esophageal hiatus hernia. Certain patients with retention of food which has become inspissated and incrusted onto the walls of the diverticulum may require repeated endoscopic aspirations to clean out the sac. This patient had to be subjected to three esophagoscopic aspirations, in addition to decompression of the esophagus by the use of a large bore tube on three occasions, in order to clean out the diverticulum. Despite this, it was impossible to remove all incrustations. This demonstrates one of the important details in the preoperative management of these patients. The esophagus can be easily emptied with the use of a large bore endoesophageal tube, as advocated by Bizzarri and Giuffrida [*24*].

Case iv. H. S., a sixty-five year old white man, was admitted to Columbus Hospital on March 9, 1954. His chief complaint was a burning sensation in the epigastrium, belching and nausea of two years' duration. For two and a half months prior to admission he had had substernal distress associated with sticking of food in the esophagus, which was more severe after nervous strain.

The hospital work-up was significant in that on barium swallow a diverticulum was found approximately 8 cm. above the diaphragm. A gastrointestinal series revealed no other abnormalities. In view of the history and x-ray findings of the presence of a diverticulum, the patient was subjected to esophagoscopy.

At esophagoscopy on March 15, 1954, the diverticulum was entered at approximately 30 cm. from the central incisor teeth. After the removal of food and mucus from the sac, it was noted that its lining was rather smooth and there was no evidence of inflammation.

On March 13, 1954, the diverticulum was excised. The operative approach and findings were similar to those in Case iii. (Figs. 4A and B.)

Postoperatively, the patient felt only slightly improved; he was discharged on March 29, 1954. He was seen once every four months. Each time he presented a history which differed slightly from that which was present prior to surgery. In view of the continuation of his complaints, the patient was readmitted to the hospital on June 10, 1956. A gastrointestinal series at this time, performed with the patient in the marked Trendelenburg position, revealed a hiatus hernia. Esophagoscopy confirmed the presence of a hiatus hernia; there was also evidence of a low grade esophagitis in the lowermost region of the esophagus. A regimen of antacids and antispasmodics was instituted consisting of gelusil® and pro-banthine.® The patient was discharged on June 16, 1956, with the diagnosis of a sliding esophageal hiatus hernia. The patient improved remarkably with this management.

Comment: It seems obvious that the symptoms in this patient were due primarily to the presence of an esophageal hiatus hernia. The incidence of symptoms due primarily to associated esophageal disease is high. Unfortunately, in this patient the associated lesion was not visualized on the first admission despite a most careful roentgenologic study. This case clearly demonstrates that when an associated lesion is found, it is mandatory to institute medical treatment for the associated lesion, if such is indicated, before surgery is advocated for the removal of the diverticulum. If there is marked improvement, no further therapy may be necessary. If not, the diverticulum and the associated lesion may both require surgery.

SUMMARY AND CONCLUSIONS

1. Pertinent data relative to epibronchial traction diverticula are presented.
2. The incidence, etiology and associated

lesions, including the clinical picture of epiphrenic diverticula, are briefly discussed.

3. The procedures that have been advocated and emphasized in the surgical treatment of epiphrenic diverticula are listed. The transthoracic approach with excision of the diverticula is the method of choice. In certain cases the presence of an associated lesion may require esophagogastrostomy.

4. Four illustrative cases of thoracic esophageal diverticula are presented, one in the epibronchial region of the traction-pulsion type and three in the lower esophageal region of the pulsion variety. Of these, one patient required no treatment of any sort; the remaining patients were treated by surgical excision.

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Reflux Esophagitis

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SINCE 1884 when MacKenzie [1] defined "oesophagitis" as "acute ideopathic inflammation of the mucous membrane of the esophagus giving rise to extreme odynophagia and often to aphagia," considerable confusion has existed in the literature as to the causes, classification and pathology of inflammatory conditions affecting the lower esophagus. Peptic ulcer of the esophagus was described by Tileston in 1906 [1] and by others who reported discrete ulcers in the lower esophagus apparently arising from ectopic gastric mucosa present in the lower esophagus. In 1929 Jackson [2] described eighty-eight cases of ulceration in the lower esophagus noted in 4,000 consecutive endoscopies, and discussed various possible etiologic factors. Winkelstein [3], however, in 1935 proposed that diffuse esophagitis in some of these cases without discrete ulceration was possibly a "peptic" esophagitis, i.e., an esophagitis resulting from the irritant action on the mucosa of free hydrochloric acid and pepsin.

Barrett in 1950 [1] differentiated between chronic peptic ulcer occurring in the esophagus which was lined by a continuous sheet of gastric mucosa from "reflux" esophagitis. The latter entity appeared to be the result of diffuse inflammatory erosion, ulceration and scarring produced by prolonged reflux of gastric juice in the presence of an incompetent cardio-esophageal sphincter and frequently associated with hiatus hernia. Whether or not scattered islets of ectopic gastric mucosa in the esophagus in contradistinction to a continuous sheet ever give rise to peptic esophagitis is debatable.

It is apparent, moreover, that the term "esophagitis" requires a clarifying adjective and that the term "peptic esophagitis" is not an inclusive term, since reflux of alkaline juices into the esophagus after surgical procedures such as esophagojejunostomy can also cause an identical type of esophagitis. Furthermore, esophagitis can also be produced by protracted vomiting, nasogastric intubation and pyloric obstruction without any pre-existing anatomic incompetence of the cardio-esophageal sphincter mechanism. It is believed, therefore, that the term "reflux esophagitis" is the more appropriate designation for this group of cases. The following factors may be active in the production of reflux and peptic esophagitis: (1) incompetence of cardio-esophageal mechanism with sliding hernia and after surgical operation; (2) gastric hypersecretion and the peptic ulcer diathesis; (3) protracted vomiting, nasogastric intubation and pyloric obstruction; (4) ectopic gastric mucosa, as "short esophagus" or scattered islets; and (5) individual susceptibility.

THE ROLE OF HIATUS HERNIA

The mechanics of esophageal hiatus hernia and its role in the production of esophagitis have been elucidated by Allison [4], Sweet [5] and others. That there is some sort of sphincter mechanism between the esophagus and stomach is generally agreed. The exact nature and mode of operation of this sphincter are still moot points. Probably there are several components to this mechanism working together in the

normal person to allow passage of food from the esophagus to the stomach and operating effectively to prevent regurgitation into the esophagus. Allison has compared the normally active diaphragmatic hiatus to a sling which aids the sphincter action of the esophagogastric junction by accentuating the obliquity of the entrance of the esophagus into the stomach. The fibers of the right crus, through which the esophagus enters, pass anteriorly around the lower end of the esophagus in a reinforced arch which pulls downward and backward on the esophagastric junction. In hiatus hernia where the weakness lies posteriorly, the stomach is allowed to slide into the mediastinum allowing the junction to rise out of this sling.

It has been stated that there are muscular fibers within the gastric wall which also act in a similar manner to that ascribed to the diaphragmatic sling [6], and that in addition there is a mucosal flap valve lying over the esophageal orifice in the normal person [7]. These latter mechanisms, it is stated, are overcome by elevating the esophagus or depressing the fundus of the stomach to eliminate the normal oblique entry of the esophagus into the stomach with the fundus rising well above this point.

Radiologic evidence tends to show what appears to be a physiologic sphincter at a point in the lower esophagus above the diaphragm.

However, the sphincter action is mediated; it does seem to exist. Even in the normal person, sufficient increase in the pressure gradient from abdomen to chest can cause regurgitation.

In the patient with disturbance of relationship of the esophagogastric junction resulting from a sliding hiatus hernia, regurgitation may occur under the abdomen-chest pressure gradient normally found, especially when this is supported or enhanced by pyloric obstruction or spasm, constriction of a herniated stomach, increase in abdominal pressure associated with obesity or merely by the assumption of the reclining position.

Further evidence of the importance of reflux as a factor in the causation of esophagitis is the high incidence of this condition following operations which destroy the normal esophagogastric relationship such as the Heyroosky-Grodahl bypassing operation, esophagojejunostomy and even after the Wendel and Heller types of cardioplasty.

Paraesophageal hernias in which the esophagocardiac relationship is not altered are not usually accompanied by esophagitis.

THE ACID-PEPTIC PROBLEM

The importance of acid-peptic regurgitation as a causative factor of esophagitis, stenosis and stricture with or without evidence of hiatus hernia has been stressed by Winklestein [8], Wangensteen [9] and others. Some confusion arises, however, as to whether the acid-peptic factor is important only when incompetence of the cardioesophageal sphincter mechanism exists or whether, in addition, a true ulcer diathesis with its associated hyperchlorhydric states, nervous tension and individual susceptibility may be present. We agree with the latter concept. The association of peptic duodenal ulceration has been high in our series (40 per cent) as well as in others. Frequently the symptoms of esophagitis may be identical to those present with duodenal ulcer. In many of our patients, impressively high free hydrochloric acid values were present. This appeared particularly significant in that esophagitis for the most part occurs in the older age groups in which decreased gastric acidity or anacidity is the common finding.

Medical measures such as parasympathetic blocking agents, antacids and sedatives, all factors which diminish the acid factor, are usually effective in relieving the symptoms of esophagitis. Finally, symptoms of esophagitis occur in only a relatively small proportion of persons with hiatus hernia. The routine coincidental demonstration of a hiatus hernia without definite symptoms is a rather frequent experience radiologically. Certainly definite reflux and bathing of the lower esophagus with gastric secretions occurs in this latter group, and the absence of esophagitis would tend to lend further strength to the importance of the acid-peptic factor as well as to the possibility of an individual susceptibility.

MacLean and Wangensteen [9] have recently reported success in the treatment of early esophageal stricture in patients by subtotal gastrectomy. Some of these patients did not have demonstrable hiatus hernias but many had gastric and duodenal ulcers. Whatever the mechanism of esophagitis leading to stricture without demonstrable reflux was in these cases, it is significant that removal of the acid-bearing portion of the stomach and promotion of rapid

drainage caused a reversal of the progression of esophagitis.

Abundant experimental evidence demonstrates the susceptibility of the esophageal mucosa to acid-peptic digestion. Selye [*10*] was able to produce hemorrhagic esophagitis in rats by pyloric ligation. Production of an esophagogastric anastomosis in dogs followed by histamine stimulation produces severe esophagitis, while removal of the acid-bearing portion of the stomach prior to anastomosis offers considerable protection against esophagitis [*11*]. Certainly, the bulk of clinical and experimental evidence indicates the great susceptibility of the esophageal mucosa to gastric juice.

DIAGNOSTIC ADJUNCTS

Refinements in diagnostic technics, particularly radiologic and endoscopic, have done much to bring the problem of hiatus hernia and peptic esophagitis to the foreground. Together they constitute the most accurate means of making the diagnosis, for symptoms of hiatus hernia and esophagitis can mimic any disease, functional and organic. Roentgenologists frequently observe hiatal hernias during an upper gastrointestinal examination as an incidental finding. In these cases, reflux is readily apparent with pressure on the barium-filled stomach during fluoroscopy. Occasionally, however, a hiatus hernia may not be readily demonstrable roentgenographically in spite of a suspected clinical diagnosis. Frequently in these cases, by placing the patient in the Trendelenburg position with pressure applied to the abdomen the herniated portion of the stomach filled with barium may be demonstrated.

The radiologic demonstration of esophagitis, however, presents a more difficult problem. In advanced cases, stenosis and stricture formation with proximal dilatation may be shown in the lower esophagus, but if one waits for these signs, the surgical treatment is usually difficult. Close inspection in early cases may reveal fuzziness of the mucosal pattern with spasm, as evidenced by lack of distensibility by a heavy barium mixture. Sometimes small areas of ulceration may be noted. The radiologist frequently is unable to tell exactly where the cardio-esophageal junction lies, the knowledge of which becomes important to the surgeon contemplating repair.

It is for these reasons that endoscopy should be an important part of the examination in suspected cases of esophagitis. The information obtained from this procedure is important from four aspects: (1) the degree of esophagitis, ulceration and stenosis can be evaluated, if present; (2) carcinoma may be ruled out by biopsy, if necessary; (3) the level of the cardio-esophageal junction can be accurately established; and (4) valuable information may be gained concerning the amount of fixation of the lower esophagus to the mediastinal structures. In long-standing cases in which fibrosis of the muscular coats and the periesophageal tissues have occurred, the lower esophagus will appear fixed by esophagoscopy. It may then be anticipated that repair of the hernia may be extremely difficult, if not impossible.

The endoscopic examination shows a characteristic set of changes. Toward the lower third of the esophagus, the mucosa may appear to be hyperemic and reddened and will frequently bleed easily on contact with the esophagoscope. The folds may be prominent, which may slightly impede the passage of the instrument. Frequently, small ulcers may be noted which at times are covered with a yellowish membrane. The cardia in these cases is lax and patulous, and the instrument passes into the stomach without deviation. This lack of deviation is diagnostic of a patulous diaphragmatic sling and is an important sign in the occasional case in which symptoms of esophagitis are present and the radiologic demonstration of an actual hernia has been unsuccessful.

OBJECTIVES OF THERAPY

In a previously reported study [*12*], a variety of surgical procedures carried out in an earlier period attested to our uncertainty in the management of reflux esophagitis but offered the opportunity of studying the effects of a number of different procedures. Further studies in these and subsequent patients have led us to plan a definite program of therapy which we believe at the present time yields the best results.

All early cases of esophagitis and those of mild or moderate degree should be given a trial of medical therapy consisting essentially of an ulcer regimen including antacids, antispasmodics, bland diet and sedatives, if necessary. Along with this program, esophageal dilatation may be indicated for mild esophageal obstruction due to edema and spasm of the lower esophagus. However, when destruction of the mucosa has occurred with progressive fibrotic

stenosis and particularly when control of symptoms by medical means has been inadequate, surgical measures are in order.

Bleeding may be a serious problem. While sudden severe exsanguinating hemorrhage is infrequent, persistent debilitating blood loss is not uncommon and calls for surgical intervention if medical therapy does not bring prompt relief.

Perforation is a dramatic event in the history of esophagitis and demands prompt surgery. However, although this complication has been reported occasionally it apparently is quite rare, and we have not seen it in our series.

While, as stated, the indications for surgical intervention in esophagitis are similar to those in duodenal ulcer, the application of surgery should be promptly made when indicated as the price of procrastination is far higher in this disease than in the case of duodenal ulcer When marked intractable stenosis of the esophagus occurs, accompanied by mediastinitis and fibrosis, the problem is far greater technically and the results poorer than in the case of pyloric obstruction due to duodenal ulcer which is treated so satisfactorily by any of several measures.

Surgical treatment of reflux esophagitis has as its first objective prevention of reflux into the esophagus by restoring as nearly as possible the normal state such as by the repair of an existing hiatus hernia. In this procedure we favor the technic of Allison. Second, rapid drainage of the esophagus is sought by relieving any obstructing condition distal to the esophagus such as pyloric obstruction. A third objective of the surgical therapy of esophagitis is the reduction of gastric acidity. Fourth, obstruction of the esophagus itself must be relieved, and in mild cases this may be accomplished by dilation after other surgery or by more direct surgical attack. A final aim of surgical therapy is the evaluation or treatment of other associated gastrointestinal disease.

TREATED SERIES

The present series of cases consists of 102 patients with reflux esophagitis treated in the past seven and a half years. Table 1 shows the operative procedures employed in this period. A total of sixty operations were performed. A few patients underwent more than one procedure. The age mode appeared in the fifth and sixth decade of life. One patient, a newborn, had esophagitis with peptic ulceration from birth. This case has been previously reported [12]. The symptoms in most cases were regurgitation of food, heartburn, retrosternal distress, dysphagia and nervousness. Hemorrhage was a prominent feature in eight patients. Severe obstructive symptoms were noted in twenty-one patients. Endoscopic examination revealed evidence of esophagitis in several cases with bizarre or minimal symptomatology. As mentioned previously, it was believed that esophagoscopy was an important diagnostic tool in these patients. Congenitally short esophagus was found in only two cases: one in a newborn with active peptic ulceration and the other in a forty-one year old woman with symptoms beginning in early childhood. It was extremely difficult in other advanced cases, however, to determine whether congenitally short esophagus was present or whether inflammation due to esophagitis with resultant scarring and contractures caused the shortening encountered. Our findings of only two cases, however, confirm the prevalent feeling that congenitally short esophagus is comparatively rare, and that it occurs in relatively younger patients usually associated with symptoms dating back to childhood.

TABLE 1

SURGICAL OPERATIONS IN PRESENT SERIES OF 102 CASES OF REFLUX ESOPHAGITIS

Gastric resection and hiatal hernia	14
Hiatal repair only	9
Vagotomy, pyloroplasty and hiatal repair	8
Vagotomy, gastroenterostomy and hiatal repair	7
Gastrectomy only	3
Esophagoplasty for stricture	6
Resection of esophageal stricture with:	
Proximal gastrectomy, esophagogastrostomy and pyloroplasty	8
Total gastrectomy and esophagojejunostomy	5
Total	60

Nine patients underwent repair of the hiatus hernia as the sole procedure when minimal or early esophagitis was present. Two of these cases, however, required further surgery, one requiring subtotal gastrectomy a year later for a bleeding duodenal ulcer. In the other case, progressive symptoms of esophagitis occurred and vagotomy with pyloroplasty was added. Gastrectomy was the sole procedure performed in three patients, all of whom had persistent symptoms which demanded subsequent repair

of the hernia. Relief was promptly obtained after repair in two patients while the third has continued to have mild to moderate dysphagia. We believe at the present time that repair of the hiatus hernia should be performed routinely, if present, in all cases in which gastric surgery is being considered for peptic disease. We do not entirely agree with Wangensteen, however, that subtotal gastrectomy may be sufficient therapy in cases in which stricturing of the esophagus has occurred from reflux of gastric content. An attempt to repair the hiatus, if defective, should be made in all cases. This, of course, may not be possible in the congenitally short esophagus or in cases in which severe stenosis and shortening of the esophagus has occurred from progressive fibrosis. Treatment in the latter type of case at best is not entirely satisfactory once stenosis and shortening with fixation of the hernia has occurred. Dilatation of the stricture in this stage usually yields only temporary benefit. Esophagoplasty was performed in six cases for relief of stricture early in the series with extremely unsatisfactory results, three cases requiring frequent dilatations postoperatively and the other three necessitating further surgery with excision of the stenosis and intrathoracic esophagogastrectomy. There is little place for esophagoplasty alone, as we see it, owing to the depth and extent of the esophageal stenosis and the likelihood of recurrent disease. Under these circumstances, we agree with Sweet [*14*] and others that the best result in these cases will be obtained by resection of the esophageal stricture along with the proximal two-thirds of the stomach followed by intrathoracic esophagogastric anastomosis and extramucosal pyloroplasty. Roux-Y esophagojejunostomy, with or without gastrectomy in this series, was attendant with multiple digestive disturbances requiring further medical care, and in a sense palliation rather than cure resulted.

Finally, the largest operative group of cases consisted of a direct attack on the acid-producing stomach along with the hiatal repair. Earlier in the series we were interested in comparing the relative merits of vagotomy and pyloroplasty, vagotomy and gastroenterostomy, and subtotal gastrectomy. Although our operative group is too small for statistical study, we now believe that repair of a sliding hiatus hernia, in cases of reflux esophagitis unrelieved by non-surgical measures, should be accompanied by subtotal gastric resection. The rationale for gastrectomy as previously mentioned is convincing. Certainly this is the procedure of choice in those cases with coexistent duodenal or gastric ulcer. In the present series, 40 per cent of the patients operated upon had direct evidence of active or chronic duodenal ulcer. The frequency of this association has been stressed by other investigators [*8,12,13*]. Moreover, gallbladder disease, pancreatitis and malignancy are frequently associated with this age group and it becomes imperative that these conditions be also diagnosed and dealt with.

To accomplish these surgical aims, therefore, we have found the most satisfactory approach to the upper abdomen and lower mediastinum is through the left thoracoabdominal incision. The patient is placed on the operating table with his left side elevated so that the plane of the back is at 45 to 60° with the plane of the table. The left arm is abducted from the trunk at a right angle and suspended from the anesthetist's shield. The appropriate eighth or ninth rib is then excised in its anterior one-half and the incision is extended across the chondral border and carried forward across the left rectus muscle. If better exposure of the duodenum is necessary, this incision can be further extended across the midline. The outer portion of the left hemidiaphragm is then incised as needed in the direction of its muscle fibers back to the tendinous portion. The left triangular ligament of the liver is sectioned and the base of the lower lobe is freed from the diaphragm if adherent. Postoperatively, the left pleural cavity is drained with a dependent tube under water seal and removed in twenty-four to forty-eight hours. Through this approach, most of the problems, foreseen and unforeseen, can most easily be dealt with and thorough abdominal exploration is readily accomplished.

In those cases of esophagitis occurring in the absence of hiatus hernia and inadequately controlled by medical measures, the most feasible surgical procedure would seem to be subtotal gastrectomy.

Finally, it should be emphasized that reflux esophagitis is a serious problem attended by a high incidence of crippling sequelae which demands assiduous study and carefully planned treatment in order to avoid the more serious complications. Even so, resistant cases remain which tax the ingenuity of the surgeon and

leave room for further improvement in the management of this disease.

SUMMARY

The etiology and clinical picture of esophagitis have been discussed.

Based on experience with 102 cases of reflux esophagitis and predicated on the etiologic factors as understood today, a program of management has been discussed.

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Peptic Esophagitis with Duodenal Ulcer

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IN 1934, I discussed before the American Medical Association a new clinical entity, peptic esophagitis, previously undescribed [1]. The association of this lower esophageal lesion with hyperchlorhydria and peptic ulcer of the duodenum or stomach led to the theory that these cases were peptic in origin, i.e., they were due to the action of pepsin and hydrochloric acid on a susceptible mucosa. Hamperl, the Viennese pathologist then delivered a paper before the Deutschen Pathologischen Gesellschaft entitled "Peptische Oesophagitis." He described the postmortem features of a group of patients with an erosive, stenosing inflammation of the lower esophagus [2]. Since these two early descriptions, many interesting papers on this subject have appeared in the literature.

Other types of esophagitis in which peptic activity (hydrochloric acid and pepsin) probably plays a prominent role are: (1) esophagitis with marginal ulceration at the cardia secondary to the hiatus hernias, associated with a short esophagus (studied by Wolf and others [3]); (2) esophagitis due to severe repeated vomiting, such as occurs frequently after abdominal operations and in pregnancy; (3) esophagitis due to prolonged esophageal intubation or frequent gastric lavages; (4) esophagitis secondary to operations in which the esophagus is anastomosed to the stomach (seen frequently after the Heyrovsky operation for cardiospasm); and (5) the solitary peptic ulcer of the lower esophagus, associated with persistent embryonic columnar epithelium.

CLINICAL FEATURES

Apparently the disease is uncommon, but Jackson has pointed out that esophagoscopies are usually performed only when there is an important indication. It is probable that in patients with gastric or duodenal ulcer who have severe heartburn and acid regurgitation, evidence of a mild esophagitis would be revealed if esophagoscopy with biopsy were employed in the study.

Symptoms. The symptoms in my series of twenty cases were dysphagia (sixteen patients), heartburn (six patients), regurgitation and/or vomiting (seven patients), substernal pain (seven patients), loss of weight (ten patients) and hemorrhage. Dysphagia was the first and most prominent symptom in seventeen of the twenty patients. Regurgitation or vomiting of sour fluid with or without food was a common symptom. Lower substernal pain on swallowing, without radiation to the back, was noted occasionally. Dysphagia seems to be the most constant and important symptom. Hyperchlorhydria occurred in 85 per cent of the cases. Three patients had a gastric acidity of 20 to 40 clinical units of free hydrochloric acid, ten patients had an acidity of 40 to 80 units, and seven had an acidity of more than 80 units.

The chief complications were stenosis, hemorrhage and perforation.

Stenosis occurred in nine patients. The role of inflammatory swelling, spasm and fibrous stricture in the production of the stenosis could be determined accurately only by a study of resected specimens or postmortem material. It is probable that all three factors are involved. Fibrous stenosing esophagitis occurs in the late stages of the severe cases.

Massive hemorrhage occurred in four patients. In three it took place after the onset of dysphagia. In all cases it took the form of hematemesis.

Perforation occurred in one patient and was found at the postmortem examination in a small hiatus hernia immediately below the area of stenotic peptic esophagitis.

Previous Operations. Four patients had undergone previous operations for duodenal

ulcer. One had had an operation for closure of a perforation, two had had gastroenterostomies, and one had had subtotal gastrectomy.

Association with Ulcer and Hernia. Fifteen patients had a duodenal ulcer and two had gastric ulcers. In one patient a gastric ulcer, and in another a duodenal ulcer, developed at a later date. One patient had a previous solitary peptic ulcer of the esophagus. In five cases a small hiatus hernia was found. These hernias are probably secondary, due to traction. In one case a larger hiatus hernia that may have been due to a pre-existing short esophagus was revealed. Paraesophageal hiatus hernias were not seen in this series.

Association with Pyloric Obstruction. It has been suggested that peptic esophagitis is prone to occur in patients with pyloric obstruction. With this in mind, a review of the cases in this series revealed the presence of pyloric obstruction before the onset of the dysphagia in only one case. Peptic esophagitis is rarely encountered, at least clinically, in large series of patients with pyloric obstruction.

RADIOGRAPHIC FEATURES

The roentgenologic findings are essentially of two varieties, namely, changes in distensibility or narrowing of the lower third of the esophagus and changes in the mucosal pattern. In severe cases the lack of distensibility may be so marked as to produce a long segment of considerable narrowing involving the lower third of the esophagus. The junction between the moderately dilated esophagus above and the narrowed portion below is gradual and symmetric. The mucosal pattern throughout the narrowed segment is distorted and may have a hazy irregular appearance. Discrete projecting ulcer craters are not seen; however, the margins of the narrowed segment may show a fine serration. Coarser irregularities are not common. The involved segment usually shows no evidence of peristaltic activity. Slight changes in caliber on repeated examinations may be noted. In the dilated segment above the involved area there may be small irregular transitory contractions. In the type of case associated with prolonged peptic esophagitis, a very small tent-like traction hiatus hernia may be seen. This occurred in five patients.

In contrast to the severe cases of peptic esophagitis described previously, persons with minimum inflammatory changes show considerably less striking findings on the roentgenologic examination. The failure of normal distensibility of the lower third of the esophagus may be seen only after the examination of numerous films, including those with the patient in the erect position. It can then be noted that this portion of the esophagus does not dilate as much as would be anticipated normally. Instead, when maximally dilated, a tubular cylindric structure with parallel side walls is visualized. Within this segment the contours of the involved portion may show a fuzziness or a fine irregularity, and the mucosal pattern again cannot be clearly delineated. When partially empty, this segment may show irregular tertiary contractions. Prompt regurgitation of barium was noted in two patients. Careful studies of this reflux are not available for the entire series. The differential diagnosis from marginal or peptic ulceration above a large hiatus hernia is not difficult. Marginal ulceration immediately above a large hiatus hernia associated with a short esophagus may be differentiated from the peptic esophagitis associated with duodenal or gastric ulcer by the following radiographic findings in cases of marginal ulceration: (1) The esophagus is short. (2) The area of narrowing in the lowermost end of the esophagus is short (1 cm. usually). (3) The esophagus above this area is pliable. (4) A slit-like ulcer crater or an ulcer patch may be identified immediately proximal to the cardia. (5) Transcardial reflux is marked and prompt. The differentiation from cardiospasm is usually simple radiographically. It may also be readily accomplished in most cases of carcinoma; however, in a small number of cases, differentiation cannot be made radiographically and esophagoscopy with biopsy is then necessary.

PATHOLOGY

Gross Pathology. The appearances on esophagoscopy are marked edema and congestion of the affected mucous membrane. This usually involves a long segment occupying the lower half or third of the esophagus, but occasionally may spread upward to involve the entire esophagus to a lesser degree. Multiple small superficial ulcerations may be seen on the surface of irregular folds. Occasionally, a larger narrow area of flat ulceration runs longitudinally immediately above the cardia on the

posterior wall. When the necrotic membrane is removed, a denuded area with an intensely red granular base becomes visible. Small nodular excrescences or a diffuse granular appearance may be present. In addition, there may be adherent white exudates in small patches or larger plaques that are readily removed by suction, exposing an edematous mucosa. Rarely, small petechiae have been observed. There is marked narrowing of the lumen in the lower esophagus that usually can be traversed by the esophagoscope, since the narrowing is most often due to spasm. In advanced cases, however, impassable organic stenosis due to fibrous stricture formation may be encountered and require repeated dilatations. In the early cases the cardia seems competent, and gastric reflux is not seen. Reflux has been observed in a few cases of long duration in which a fibrous replacement of the cardia has occurred.

Microscopic Pathology. The microscopic features have been presented in considerable detail by Hamperl. He mentions the following as early lesions: epithelial necrosis, epithelial hyperplasia, a hyaline mucosal zone splitting the superficial epithelial layer, polynuclear infiltration, and hypertrophy of the muscularis mucosae. These changes are generally accepted by pathologists as evidence of peptic inflammation, i.e., inflammation due to the destructive action of hydrochloric acid and pepsin on a susceptible mucosa, and form perhaps the best diagnostic criteria of peptic esophagitis. Finally, fibrosis and stenosis are seen. The erosions, as a rule, do not penetrate through the muscularis mucosae. This diffuse, superficial ulceration seen in peptic esophagitis does not, in my experience, eventuate in the deep, circumscribed solitary peptic ulcer of the lower esophagus.

THERAPY

Medical Therapy. All of these patients were given the conventional Sippy type of therapy with anticholinergic drugs, including methantheline bromide (banthine®) in some cases, and alkalis. Six patients were treated with continuous intraesophageal milk-soda drip therapy. Mechanical dilatation was employed in six cases. This was usually started by passing graded dilators through the endoscope. When a No. 36 French dilator could be passed, endoscopic dilatation was discontinued and a No. 36 French Hurst mercury-weighted rubber esophageal bougie was substituted. The size of the tube is gradually increased if possible to a No. 40 French. The results of this type of therapy are often remarkably good. These medical measures are usually successful and result in either a cure or a remission. I have seen a markedly constricted esophagus widen to almost normal diameter after dilatation.

Surgical Therapy. One patient underwent transthoracic bilateral vagotomy and four patients underwent subtotal gastrectomy. In general, the results of these operations have been disappointing. This may be due to the fact that surgery was instituted only in the most severe cases with marked stenosis, hemorrhage or intractable dysphagia. Whether the poor results are due to the persistence of free acid or to a continuation of the disease process into greater stenosis is conjectural. The fact remains that subsequent dilatations were necessary to relieve these patients from progressive stenosis. Probably the surgical procedure of resection of the lower esophagus and upper half of the stomach with bilateral vagotomy and esophagogastrostomy seems best.

COURSE AND DIAGNOSIS

Sixteen patients seemed to have done well with medical or surgical therapy. Three still had symptoms despite therapy, and one died of a perforation.

The most important disease to be differentiated is, of course, carcinoma of the lower esophagus. The occurrence of peptic esophagitis in the older age group, the type of symptoms, the marked loss of weight and the radiographic features combine to suggest carcinoma strongly. Although the radiographic features of peptic esophagitis are characteristic, they may be imitated exactly in a few rare cases by a carcinoma. Endoscopy alone may also be inadequate for the differentiation; therefore, biopsy is imperatively indicated.

Cardiospasm is usually easily differentiated radiographically and endoscopically. Other types of esophagitis may be diagnosed and separated from this group by the history and observations of definite causal agents. Such agents include prolonged vomiting, intubation, extensive burns, general infections, cerebral lesions and chemical agents. I have also encountered a few cases of peptic esophagitis

without any apparent etiologic explanation. These will perhaps be clarified by the occurrence of a peptic ulcer of the duodenum or stomach later in the history.

SUMMARY

This disease is apparently one that afflicts men in the older age groups. In the group under consideration, duodenal ulcer or gastric ulcer (and in one case a previous peptic ulcer of the esophagus) were constantly associated. From this feature, the location of the lesion in the lower third of the esophagus and the hyperchlorhydria in most cases, the conclusion seems inescapable that this disease is peptic in origin. The response to medical and surgical antiulcer therapy is striking.

What the ultimate, detailed mechanism of peptic esophagitis is seems purely conjectural at present. Reflux may result from the hypersecretion associated with duodenal ulcer or from an unexplained decrease in the tone of the cardial sphincter. Perhaps the characteristic nocturnal hypersecretion with the patient in the horizontal position leads to a prolonged bathing of the lower esophagus by the highly acid gastric contents. As a result, localized peptic esophagitis occurs in the lower esophagus with a spastic narrowing.

The disease is characterized by a long history of duodenal or gastric ulcer with a subsequent shorter history of esophageal symptoms. The chief symptoms are dysphagia, heartburn, regurgitation, substernal pain and loss of weight. The complications are stenosis, hemorrhage and perforation. There is no evidence that this is a precancerous disease. The response to medical therapy and mechanical dilatation is usually good. In a small percentage of the patients, surgical therapy is necessary.

This condition is important not only as a disease entity but also in the differential diagnosis from carcinoma of the lower end of the esophagus.

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Peptic Stenosis of Esophagus

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BENIGN stenosis of the esophagus is a general term which means narrowing of the esophagus, acute or chronic, due to acid, peptic regurgitation from the stomach. We are excluding web, lye stricture, and achalasia since they are separate diseases unrelated to the stenosis of peptic esophagitis or ulceration.

Incidence. Increasing interest in esophageal disease coupled with improved methods of radiologic and endoscopic diagnosis has resulted in greater awareness of this condition.

In 1931 Vinson [1] analyzed 243 cases of esophageal obstruction. Forty were of unknown origin. We believe many of these represented stenosis secondary to peptic esophagitis or ulceration. More recently Benedict and Gillespie [2], in a study of 1,340 patients examined in this clinic, found 119 (8.7 per cent) cases of peptic stenosis. Lodge [3] found microscopic evidence of acute esophagitis in 31 per cent of 500 unselected hospital patients and in 3 per cent of 100 patients dying acutely in accidents.

This disease appears to be slightly more common in males than in females, in the proportion of 60: 40 in our series. The age of onset is usually between forty and seventy years, with extremes of fifteen and ninety years.

Etiology. In all cases of benign stenosis there is associated esophagitis. We believe the esophagitis precedes the development of the stenosis and is an important factor in the etiology [4]. Regurgitation of acid, peptic gastric secretions probably plays a role in the development of esophagitis and esophageal ulcer. Hiatus hernia is probably the most common single predisposing cause. In our series 85 per cent of the patients had a hiatus hernia. Forty per cent showed evidence of esophageal ulcer on roentgenologic examination. Forty per cent also showed roentgenologic evidence of present or past duodenal ulceration. One hundred nineteen biopsy specimens were taken from eighty patients. Forty-nine of these showed gastric mucosa in one or more biopsies (62 per cent); the remainder showed acute and chronic inflammation of the esophageal mucosa.

As early as 1906 Tileston [5] held that insufficiency of the cardia allowing regurgitation of gastric juice was essential for the development of the esophageal ulcer and hence that vomiting—as in peritonitis, duodenal ulcer and pyloric stenosis—was of importance. He reported three new cases of esophageal peptic ulcer and stated that the scarring of these large esophageal ulcers resulted in esophageal stenosis.

Winkelstein [6] in 1935 described five cases of a new clinical entity called "peptic esophagitis" and held that the course of the disease, the location of the ulcers, the association with peptic ulcers, the hyperchlorhydria and the relief after ulcer treatment suggested peptic origin of the esophagitis.

Allison [7], in a significant paper on seventy-four cases of peptic ulcer of the esophagus, considered hiatus hernia allowing regurgitation of acid gastric juice the essential factor. Olsen [8] regarded reflux of gastric juice as the most common cause of esophagitis, and stressed the influence of vomiting and incompetence of the cardia from such conditions as hiatus hernia. Benedict and Sweet [9], in a report of sixty cases of benign stenosis of the esophagus, held that regurgitation of acid gastric secretions was probably an important cause of esophagitis and benign stenosis.

Experimental Evidence. In 1938 Selye [10] described the production of what he called hemorrhagic esophagitis by pyloric ligation in rats, indicating that the presence of gastric juice for an abnormal time in a normal esophagus would produce inflammation.

Ferguson et al. [11], in experiments on various types of animals, found that acid gastric juice in contact with the esophagus for an abnormal time, whether from vomiting or regurgitation or by direct application, had a "devastating effect" whereas hydrochloric acid in similar concentration to that in gastric juice had little influence by itself. They believe that "non-specific esophagitis is due primarily to the action of acid-peptic juice."

Pathology. The pathologic changes are most severe distally since the injurious agent arises from below.

In mild cases there may be only mucosal edema with hyperemia. This may progress to mucosal erosion and involvement of the submucosa. Healing may occur without stenosis and shortening. In more severe cases the entire mucosal layer may be stripped away, revealing patches of inflamed muscle. Perforation may occur. The cicatricial healing of these changes results in esophageal shortening and stricture.

The interested reader is referred to the excellent review of Peters [12] for the detailed pathology of this condition.

Symptomatology. The outstanding symptom of benign stenosis of the esophagus is dysphagia, varying from slight difficulty with solid foods to complete inability to swallow any liquids at all. Regurgitation is common, usually without nausea and without real vomiting. Heartburn is frequently encountered and there may be substernal distress or sub-xiphoid pain, especially if there is ulceration. Anorexia is also common; there may be inability to belch. The regurgitated material may be bloody if inflammation is severe, and hematemesis or melena may occur with major ulceration.

DIAGNOSIS

The diagnosis of benign peptic esophageal stenosis may be suspected if there is a history of dysphagia, substernal pain and regurgitation. There may be a history of symptomatic relief by a bland liquid and semi-solid diet. X-ray examination is of very great importance and typically reveals a smooth, funnel-shaped narrowing of varying length through which the barium passes slowly if there is very much obstruction. With little obstruction, however, there is not much delay in the passage of barium. Unfortunately, the radiologist may be unable to differentiate benign stenosis from carcinoma. In all cases, esophagoscopy is of great importance.

Esophagoscopy. Direct inspection of benign, inflammatory esophageal stenosis usually reveals a smooth, conical narrowing with varying degrees of inflammation. The degree of inflammation is quite unpredictable from the symptomatology and x-ray findings. Usually there is a good deal of reddening, edema, and often erosions and superficial ulcerations. Esophagitis may be marked and may extend 10 or 15 cm. above the area of narrowing. Not infrequently a vegetable foreign body or a small bolus of meat may be found lodged in the site of the stenosis. Such foreign material should be removed, bouginage carried out and a biopsy specimen taken in order to exclude carcinoma. The site of the biopsy is of very great importance. Of course the specimen must be taken with great care to avoid perforation and it should usually be taken from deep within the lumen of the stenosis in order to get a typical area. It is possible, however, to introduce the forceps too deeply beyond a stenosed area and get a benign report of inflammation just below an area of carcinoma. In our experience, however, this has been a rare occurrence. If carcinoma is still suspected after a biopsy showing only chronic inflammation, esophagoscopy and biopsy should be repeated.

Cytology. In recent years cytologic study of esophageal washings has become important, and in rare cases this method has established a positive diagnosis of carcinoma when other methods have been doubtful or negative. However, one must still beware of false positive examinations.

TREATMENT

Dietary. The esophagus is continually subject to trauma by the passage of food and drink. Unwise eating or drinking may be very injurious to normal esophageal mucosa and is certainly of no benefit to an already inflamed mucous membrane. Some patients with severe inflammatory stenosis of the esophagus should be treated first by intravenous therapy and given nothing by mouth. Later, bland liquids and thin cereals may be tolerated. Fruit juices may be irritating. When the narrowing is not too marked, strained vegetables and ground meat may be tolerated. Alcohol, fried or highly seasoned foods should be entirely omitted.

Medicinal Therapy. Antacids are useful in the treatment of peptic esophagitis with or without stenosis. Aluminum hydroxide is excellent either in liquid or tablet form such as amphojel,® creamalin® or gelusil.® Sodium bicarbonate is also useful but cannot be used over a prolonged period because of the danger of formation of kidney stones and metabolic imbalance. Occasionally there is an element of spasm in inflammatory diseases of the esophagus which may be benefited by the use of nitroglycerine, $\frac{1}{200}$ gr. dry under the tongue just before a meal. Atropine usually has little effect on esophageal spasm.

Bouginage. Except in certain intractable cases the treatment of benign stenosis of the esophagus is bouginage. Esophagoscopy should always be carried out in every case of benign stenosis in order to observe the degree of inflammation and stenosis present, and also to carry out bouginage under direct vision and to obtain a satisfactory biopsy specimen. When carcinoma has been excluded and the diagnosis of benign peptic stenosis established, further bouginage can be carried out in the office or in the outpatient department using a previously swallowed thread as a guide. The patient is given a No. 1 silk thread and instructed to swallow it slowly, at a rate not exceeding 6 inches per hour, until it is well anchored in the intestinal tract. If it is swallowed too rapidly, it becomes knotted and cannot be used as a guide for bouginage. Instructions for swallowing of thread are as follows [4]:

1. Fasten a large spool of No. 1 white silk thread (obtained from a surgical supply house) on the patient's clothing or in the upper breast pocket.

2. Instruct the patient to take the end of the thread in his mouth and swallow it with water or any liquids or foods. It sometimes helps to attach the thread to a gumdrop or a lifesaver.

3. Do not put any lead shot on the end of the thread.

4. Do not tie knots anywhere in the thread except at the end for the purpose of fastening it to a gumdrop or lifesaver.

5. Instruct the patient to swallow the thread slowly—at a rate no faster than 6 inches per hour. Otherwise it will get tangled up in the esophagus. Do not awaken the patient at night for purposes of swallowing thread.

6. Cut the thread at the anus when it appears with the bowel movement.

7. Do not cut the thread between the spool and the mouth.

8. Do not keep pulling the thread at the mouth to see if it is anchored. It usually takes two or three days to get it firmly anchored.

9. For purposes of measurement mark the thread with one ink mark at 5 feet, two ink marks at 10 feet, three ink marks at 15 feet and four ink marks at 20 feet.

Bouginage is then performed. Bougie olives of varying sizes are attached to the flexible tip through which is threaded the No. 1 white silk thread, and by pulling the thread taut at the patient's mouth the bougie is safely passed the full length of the esophagus dilating the narrowed area. It is advisable not to pass more than three bougies at one treatment because of the danger of perforation. If there is much resistance to the passage of the bougie, a smaller size should be used. Bouginage should be temporarily discontinued if there is much bleeding. When these rules have been observed, this type of bouginage has been 100 per cent safe in our hands. Most patients who can be dilated to No. 35 French can eat everything satisfactorily provided they eat slowly and chew their food carefully. Even if the food has to be mashed or ground, some elderly poor-risk patients prefer to continue with bouginage rather than to consider surgery. In young good-risk patients, however, when there is a great deal of fibrosis and poor response to bouginage, surgical therapy should be considered. There are also some patients who have had severe hemorrhage from esophageal ulcer; in such patients surgery is also indicated.

Surgical Treatment. The indications for surgery are hemorrhage or failure of conservative management. Minor bleeding episodes are usually self-limited and respond well to medical management. Massive hemorrhage or chronic bleeding associated with anemia requires resection. The point at which bouginage fails is often difficult to define. The older poor-risk patient may get along indefinitely on a regimen of soft food and periodic dilatations—a program that might not be acceptable to a younger, more active individual.

Patients with long established esophageal stricture and irreversible fibrosis frequently have a coexisting duodenal ulcer with some pyloric obstruction. It is frequently possible to remedy both conditions by performing pyloroplasty in addition to the esophageal resection.

Rarely one may encounter sharply localized stricture or areas of stenosis which do not respond to bouginage and are amenable to local procedures. Esophagoplasty consisting of vertical incision through all layers and careful transverse closure is sometimes admirably suited. When the extent of narrowing is well localized but too extensive for esophagoplasty, local resection with end-to-end anastomosis can be performed. It must be remembered, however, that several centimeters of normal esophagus must be mobilized at either end in this procedure, and meticulous care and judgment must be used so as not to jeopardize the blood supply to the anastomosis.

Most esophageal strictures requiring surgery necessitate esophageal resection and esophagogastric anastomosis. We have utilized a left transthoracic approach. The diaphragm is divided through the esophageal hiatus and the stomach is mobilized. Great care must be taken to identify and preserve the right gastric and right gastroepiploic vessels. The esophageal dissection may be bloody and difficult because of local inflammatory reaction. The esophagus should be transected well up through normal tissue since microscopic inflammation may extend well beyond any visible changes. Failure to remove all the diseased tissue may result in restenosis. Not infrequently a supra-aortic anastomosis will be required. Postoperative esophagitis is a less frequent complication in the higher resections. It is possible that the greater number of mucous glands present in the upper esophagus provide a greater degree of protection.

Adequate gastric resection is also necessary in order to obtain maximum reduction of acid secretion. Most of the lesser curvature and all the body and fundus of the stomach are excised, forming a tube of the greater curvature as described by Sweet [*13*]. Concomitant pyloroplasty is usually advisable because of the gastric atony resulting from the vagotomy of esophageal resection as well as the high incidence of duodenal stenosis associated with benign stenosis.

PROGNOSIS

The prognosis in benign stenosis of the esophagus is very good as regards palliation by bouginage. Complete cure by bouginage probably occurs very infrequently because some narrowing almost always remains, as can be demonstrated by x-ray examination. Clinically, however, a good many patients are able to eat practically everything after a series of bouginage treatments. Surgery when carried out with meticulous care, especially as regards the anastomosis, usually gives good results; however, there have been some recurrences of inflammatory stenosis at the esophagogastric anastomosis. In such cases further bouginage may be necessary.

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Corrosive Esophagitis

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THE swallowing of caustic agents such as acids and alkalis will, in the majority of cases, produce burns in the esophagus severe enough to result in strictures. These strictures may seriously impair the function of the organ or even render it useless. Patients so afflicted present a problem in care, management and rehabilitation which would be difficult to overstate. Prolonged hospitalization, difficult mechanical and surgical procedures, and years of follow-up spell out the cost in time, money and suffering.

A great deal has been accomplished toward prevention by the laws requiring that these agents be labeled "poison" and by educating the public to their dangers. However, accidental burns of the esophagus occur all too frequently in areas where lye is used as a household cleaning agent. In the adult population, the greatest number of cases of esophageal burns are the result of attempts at suicide.

The object of treatment of the burned esophagus is to guide the healing processes so that the swallowing function of the organ is preserved. It should be based on a clear understanding of the pathologic changes that are taking place in the organ.

PATHOLOGY

The severity of the lesion depends upon the corrosive agent swallowed, its nature, concentration, quantity and the duration of its contact with the tissues.

Corrosive Agents. The majority of patients with corrosive esophagitis admitted to Bellevue Hospital had taken the corrosive agent in an attempt at suicide. Almost anything with lethal possibilities was swallowed. Among these were lye, Drano, washing soda, phenol, Lysol, formaldehyde, ammonia, smelling salts, Clorox, sulfuric acid, nitric acid, lactic acid, potassium permanganate, bichloride of mercury and iodine. Clinitest® tablets used in urine tests in the home have accidently been swallowed and have caused severe burns of the esophagus.

Chemical Nature of the Agent. Acids and alkalis are most destructive to living tissues. Sodium and potassium hydroxide and ammonia produce the most severe burns and account for more than half of all corrosive strictures occurring in the esophagus.

Alkalis have the ability to penetrate to the deeper layers of the esophagus, because they produce a liquefying necrosis. All the layers become involved in an intense inflammatory reaction.

In contrast, acids produce a coagulative necrosis of the superficial layers of the esophagus. A firm eschar forms which limits the penetration of the agent and scarring is less extensive.

Lysol was taken frequently by the patients in this series. It produces a superficial esophagitis that does not extend to the deep layers. Healing is rapid and, in my experience, completed without stricture formation. Although strictures have been reported as occurring after ingestion of Lysol, we have not seen a single instance and have concluded that it must be exceedingly rare. The importance of this was brought to our attention recently by a patient who had undergone gastrostomy shortly after he had taken Lysol. Esophagoscopy did not reveal any lesion in the esophagus and it is certain that the operation could have been avoided if it had been realized that Lysol would not produce a stricture.

Iodine usually produces extensive superficial lesions of the esophagus, many of which will heal without impairing the function of the organ. However, we have seen two patients in whom severe strictures developed.

Concentration of the Agent. Even small quantities of a concentrated caustic, taken into the mouth and then expectorated, may produce lesions in the esophagus as the agent mixes with the saliva and is swallowed. The more concentrated the agent, the more severe the injury will be, both in depth and extent. The experimental studies of Krey [1] show that there is a definite quantitative relationship between the depth of penetration of the lye and its concentration, as follows:

Normal (3.8 per cent) solution of sodium hydroxide with a ten-second contact with esophageal mucosa produced a necrosis of the mucosa and submucosa and involved some fibers in the inner longitudinal layer in rabbits. 3N (10.7 per cent) solution of sodium hydroxide produced necrosis extending to the circular muscle. 5N (16.4 per cent) sodium hydroxide produced necrosis extending to the outer longitudinal muscle. 7N (22.5 per cent) sodium hydroxide produced necrosis extending throughout the whole esophageal wall and also affected, to some extent, the periesophageal tissues.

The significance of these figures is obvious when one realizes that cleansing and washing powders have from 8 to 50 per cent caustic soda as the chief ingredient.

Quantity and Duration of Contact. Larger quantities of corrosive substances are swallowed by persons attempting suicide or who are in an alcoholic stupor. The lesions are more extensive in these cases and the injury also involves the stomach and duodenum. Duration of contact will also determine the depth of penetration.

Histologic Changes. Belinoff [2], from a study of experimentally burned dogs' esophagi, divided the course of corrosive lesions into four stages: (1) necrosis, (2) ulceration, (3) granulation and (4) scar formation.

In the acute phase, lasting two weeks, death of the cells takes place by coagulation of the intracellular proteins. A zone of intense inflammatory reaction develops in the living tissue surrounding the area of necrosis. The necrotic tissue sloughs, leaving an ulcerating surface with an acutely inflamed base. All the layers of the esophagus are usually involved in the inflammatory reaction.

Grossly, areas of ulceration and slough are found in the oropharynx, epiglottis, posterior surface of arytenoids, at the cricopharyngeus and the crossing of the left main bronchus and cardia. In this group of patients the most severe burns occurred at the cardia and middle third of the esophagus.

By the end of the first week all the coats of the esophagus are involved with inflammatory edema and the organ becomes thick and semirigid. New blood vessels form and fibroblasts proliferate. By the end of the second week granulation tissue is replacing the defects of the sloughing areas. The fibroblasts lay down collagen fibers.

At the beginning of the third week the collagen fibers begin to contract, causing narrowing of the esophagus. Adhesions form between the granulating areas. Pockets and bands develop. The inflammatory reaction involves the muscular coats and also the myenteric plexuses of nerves, both Meissner's and Auerbach's plexus. Degenerative changes take place in both the muscle and nerve tissues. Finally, healing is accomplished after months of inflammatory reaction by the replacement of the muscularis mucosa and muscular coats with dense fibrous tissue and the replacement of the epithelium with a low stratified squamous epithelium, devoid of glands. Grossly, the esophageal lumen is narrowed, is made up of multiple channels and pockets or may be completely occluded.

The histologic picture and also the clinical picture will vary with the severity of the burn and the depth to which the corrosive agent penetrated. Slight burns will heal without producing any changes in the mucous membrane of the esophagus. Deeper burns involving the muscle layer will produce greater scarring and when the necrosis extends throughout the entire wall, periesophagitis, pleurisy, pericarditis and even mediastinitis may develop. Perforation of the necrotic wall may involve the left main bronchus, producing a bronchoesophageal fistula.

CLINICAL PICTURE

The clinical course varies according to the severity of the esophagitis and patients may be grouped into three categories: (1) mild esophagitis, (2) severe esophagitis without complications, and (3) severe esophagitis with complications.

Mild Esophagitis. The mild cases are those which are produced by weak acids and alkalis. Soreness of the mouth and slight dysphagia are present.

Severe, Uncomplicated Esophagitis. This

form is produced by the strong acids and alkalis and is almost invariably present after the ingestion of lye. Soreness of the mouth and severe dysphagia, nausea and vomiting, and a rise in temperature are the common symptoms. These account for the greatest number of cases and the clinical picture is due solely to the injury of the esophagus.

The clinical course of the uncomplicated severely burned esophagus may be divided into three phases: (1) the acute phase, (2) the intermediate or latent phase, and (3) the chronic or obstructive phase.

The acute phase lasts for about three days and is characterized by pain, dysphagia and a slight rise in temperature.

The intermediate or latent phase lasts two to six weeks and is characterized by gradual improvement and disappearance of symptoms. This apparent favorable clinical course frequently leads one to believe that all is going well in the esophagus.

The chronic or obstructive phase begins about the third to sixth week and its onset is marked by the return of dysphagia, which may be progressive until the esophagus becomes completely obstructed. This corresponds to the time of contraction of the collagen fibers.

An interesting observation is the disturbance in the physiology which ushers in the obstructive phase. Between the third and sixth week the patient experiences difficulty in swallowing and in the course of a few days he may be unable to take even liquids. Esophagoscopy at this time will reveal that the lumen of the organ is still present, although considerably narrowed. The esophageal wall can be observed contracting "*en masse*." The cause of the obstruction is not organic but physiologic. The burned esophagus reacts to stimuli of food with a tonic spasm, thus the normal physiologic mechanism of propelling food through the esophagus is disrupted and the patient regurgitates. The practical significance of this is that a string may be passed at this time and a lumen preserved for future dilatations.

Severe Esophagitis with Complications. When the injury to the esophagus is severe and extends to the periesophageal structures, or is extensive and involves the gastrointestinal tract, the clinical picture is more grave and the presenting signs and symptoms are those of the complications. Mediastinitis, esophageal perforation, pericarditis, chemical gastritis and peritonitis, pulmonary edema and laryngeal obstruction are some of the early severe complications. The prognosis in this group is poor and the mortality rate high.

DIAGNOSIS

The history of swallowing a caustic agent or signs of burns about the mouth and lips may be accepted as presumptive evidence that a burn of the esophagus has occurred. However, it is necessary to establish definitely whether or not such a lesion does exist before starting the patient on any formidable method of treatment or before discharging him.

Moreover, it is important to realize that it is often impossible to correlate the findings in the mouth and pharynx with the esophageal changes. Thus, a patient may have severe burns of the mouth and pharynx without showing any evidence of burns in the esophagus. Conversely, and much more important, burns may be absent in the mouth and pharynx and yet a severe burn of the esophagus may be present.

One of the most common errors in diagnosis is the result of observing the improvement that takes place in the latent phase. One is often led to believe that if the patient has less difficulty swallowing and is feeling better, the esophagus must also be improving. This is usually not true and time, important in treatment, is lost.

The methods of examination that are available to us are x-ray studies of the esophagus and esophagoscopy.

X-ray studies of the esophagus, if carefully done, will indicate early mucosal changes and lack of motility. However, esophagoscopy has proved to be the most accurate and informative means that we have at our disposal. It permits direct examination of the extent and severity of the burn. It is also a means of following the progress of healing. Although the procedure is potentially dangerous, if carried out with care, injury to the organ can be avoided. It is our policy to perform esophagoscopy in every patient who gives a history of having taken a corrosive agent. If the patient is a child, or an adult unable to cooperate, esophagoscopy is done under general anesthesia. The esophagoscope is passed to the first burned area, but not beyond it. In this way the possibility of perforation can be avoided.

Having thus directly established the presence of a burn, its extent and severity, one can plan the management and also arrive at a prognosis.

TREATMENT

Treatment will depend upon the severity of the burn, the systemic reaction to the injury and the development of complications.

These measures are directed toward: (1) controlling shock, restoring electrolytic balance, maintaining nutrition and counteracting secondary infection; (2) the management of complications such as gastrointestinal perforation, peritonitis, mediastinitis and laryngeal obstruction; and (3) the management of the esophageal lesion itself.

Since there is a wide variation in the clinical picture each case must be studied individually. In many instances the general management of the patient and the complications will take precedence over other measures directed toward the esophagus.

Each of the clinical stages of corrosive esophagitis has its particular problem: (1) neutralization of the agent in the acute phase; (2) prevention of strictures in the latent phase; and (3) management of strictures in the obstructive phase.

First Stage. Dilute acetic acid (2 per cent) or half-strength vinegar has been used to neutralize alkalis and sodium bicarbonate for acids. Unless these are taken immediately, they are of very little value. Kiviranta [3] could find no evidence that neutralization, even when done within minutes after ingestion, altered the course of the patient. Certainly these measures can be eliminated when there is a delay of several hours before the patient presents himself for medical care.

If the patient is dehydrated, fluids should be given intravenously and the electrolytic balance restored.

Peroral feeding should be withheld during the period of nausea, vomiting and dysphagia.

Second Stage. Since the burned esophagus begins to narrow sometime during the second to third week, measures aimed at preventing strictures must be started immediately.

The granulation tissue reaction to the initial injury by the caustic agent is undoubtedly maintained and increased by secondary infection. This factor can be controlled by the administration of 1 million units of penicillin and 1 gm. of streptomycin per day. Penicillin suspension taken by mouth also has a definite beneficial local effect.

The small polyethylene feeding tube can safely be used in the esophagus for maintaining nutrition of the patient. The tube is non-irritating and its small size does not produce pressure points. In this way, the esophagus is put at rest. It also diminishes the amount of granulation tissue produced by food particles becoming enmeshed in the tissue and acting as foreign bodies, as has been shown by Krey [1] in the experimental animal.

The corticosteroids and ACTH, because of their inhibiting effects on granulation tissue production and their anti-inflammatory action, are effective therapeutic agents in the management of corrosive esophagitis.

Rosenberg et al. [4] reported success in controlling corrosive esophagitis in animals treated with cortisone. Since that time, there have been many reports in the literature [5–7], some of clinical success and others of failure of cortisone to prevent strictures. However, the steroids must not be though of as specific preventatives. Strictures have occurred in several of our patients while on cortisone therapy. This has also been reported by others. In severe gastric burns, cortisone should not be used because of the danger of perforation. The dosage recommended for cortisone therapy is 400 mg. the first day, 300 mg. the second day and then gradually reducing the dosage until 50 mg. per day is given. This should be maintained for four to six weeks; or the prednisteroids, prednisone or prednisolone, may be used. The principles of corticosteroid therapy should be followed here as elsewhere.

Salzer [8], in 1920, was the first to advocate early dilatation as a means of preventing strictures by directing the formation and contraction of scar tissue so that the lumen was maintained and adhesions prevented. In 1924, Bokay [9] reported success in 117 of 132 cases treated with prophylactic bouginage. In this country, Martin and Arena [10], Gellis and Holt [11] and Blassingame et al. [12] have reported good results using this method.

The mercury-filled bougies (Hurst) are passed each day, usually starting within the first four days after the burn. The bougies are allowed to drop into the esophagus by their own weight. In adults, dilatation is continued to a No. 40 French or above; in children, a No. 30 French or above. The largest bougie passed each day is left in place for twenty minutes, or as long as the patient will tolerate it. The dilatations are continued as long as

there is a tendency of the scar tissue to contract down. The average period of daily dilatations is one month. After this, the frequency of dilatation is diminished progressively. If, after four weeks, the patient requires further dilatation, he can be provided with a bougie to be used at home.

Results of prophylactic dilatations have been good in the hands of most observers [*13*]. Success can be judged by the degree of swallowing function maintained and the size of the lumen, as measured by the largest bougie that can be passed. An esophageal lumen which will accept only a No. 30 French bougie or less is considered unsatisfactory, because the patient can only manage semi-solid foods. A No. 40 French bougie, or higher, is considered satisfactory.

Third Stage. In the third stage the recurrence of dysphagia indicates that the stricture has formed. The following methods of dilating strictures have been used: (1) Dilatation from above with the Jackson bougie under direct vision through the esophagoscope. This method is ideal for single strictures. When multiple strictures are present, it is extremely hazardous because the bougie may perforate through a pocket formed by adhesions beyond the point of vision. (2) Dilatation from above over a string swallowed by the patient. This method eliminates the dangers of blind dilatation in multiple strictures. It does not require gastrostomy. This has the added advantage of simplifying the task of the surgeon if gastroesophageal anastomosis is done at a later date. (3) Retrograde dilatation. This is the safest and most successful method of dilatation of severe extensive strictures. Dilatations are carried out weekly and may require one year or more. Gastrostomy is necessary. Recently, Tucker [*14*] has modified his bougie. These are now cannulated so that they can remain in place for twelve to twenty-four hours. The danger of decubitus ulceration developing, due to leaving the bougie *in situ* for this period, has to be kept in mind.

SURGERY OF THE ESOPHAGUS IN CORROSIVE ESOPHAGITIS

The great strides that have been made in thoracic surgery have naturally prompted the question of what part surgery should play in the management of strictures of the esophagus due to corrosive esophagitis.

Resection of the esophagus and anastomosis with the stomach or jejunum would have much in its favor. It would eliminate the prolonged period of dilatation through a gastrostomy and its many inconveniences. However, there are several difficulties which make this operation undesirable as the routine method of handling strictures of the esophagus. Resection of a severely burned esophagus, which has been involved with a periesophagitis, is technically difficult. This difficulty is further increased by the fact that the burns may involve the upper portion of the esophagus, requiring the surgeon to make the anastomosis at a point involved with scar tissue. The possibility of stricture formation at the point of anastomosis has not been eliminated. The consensus of thoracic surgeons is that transplantation of the stomach into the thorax is not well tolerated by children. It would seem, then, that the measures previously outlined should be used first before surgical intervention is decided upon.

Indications for Surgery. These are as follows: (1) an esophagus with complete stenosis, in which all attempts from above and below have failed to establish a lumen; (2) an esophagus with marked irregularity and pocketing; (3) an esophagus which cannot be dilated without the development of a severe periesophageal reaction or mediastinitis; (4) an esophagus with fistulas; and (5) an esophagus which cannot be dilated or maintained above a No. 40 French lumen. Also included are those cases in which the patient is unable, or unwilling, to undergo the prolonged period of dilatation.

Sweet [*15*] and others have reported successful resection of the esophagus and anastomosis with the stomach.

CONCLUSIONS

1. Management of corrosive esophagitis should be based on a knowledge of the pathologic changes that occur in the organ.
2. Measures directed toward the prevention of the strictures should be started early to be effective.
3. Cortisone and antibiotics reduce the local reaction in the esophagus and indirectly reduce the tendency to stricture formation.
4. In the majority of cases the function of the esophagus can be maintained by conservative methods.
5. Early dilatation with weighted bougies

maintains the lumen and prevents adhesions between ulcerated areas in the esophageal wall.

6. When strictures have developed they can be successfully managed either with peroral or retrograde bouginage through a gastrostomy. It is important to realize that each case presents problems which require individual consideration. With the study of each patient and the application of the numerous technics for mechanical dilatation, the majority of esophagi can be restored to good functioning condition.

7. Strictures with complicated adhesions, irregularities or periesophageal reactions, fistulas or complete stenosis are better treated with surgery.

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Lower Esophageal Web

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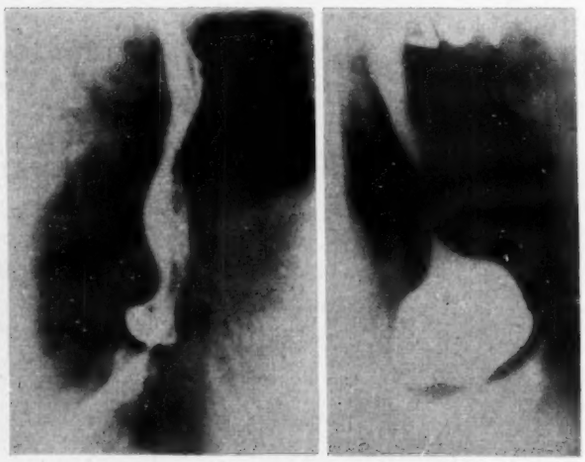
LOWER esophageal lesions causing dysphagia usually are diagnosed accurately by barium swallow esophagogram and esophagoscopy with direct visual inspection and/or biopsy. Some common obstructing lesions are carcinoma, cardiospasm, stricture or stenosis, diverticulum, and an entity recently described by the author called lower esophageal web.

In 1953 Ingelfinger and Kramer [1] published the case histories of six patients with dysphagia. All had similar x-ray findings showing a constriction in the barium column at the lower end of the esophagus which the authors called "contractile esophageal ring." In the same year Schatzki and Gary [2] reported five cases in which there were similar roentgenographic findings which they called "lower esophageal ring." Each author described the radiologic findings in accurate detail and then each applied a new nomenclature to describe his concept of this lesion. Each group had one case in which treatment was surgical (without definitive pathologic diagnosis). Each of these authors was of the opinion that the lesion could be recognized as a radiologic and functional entity, the pathology of which was as yet uncertain.

Bugden and Delmonico [3] reported two cases of "lower esophageal web" meeting the radiologic criteria described by Ingelfinger and Kramer, and Schatzki and Gary. These two patients were subjected to operation. Unfortunately, in only one case was there a pathologic report on the specimen. Each patient suffered with severe, painful dysphagia and presented the typical x-ray findings described by the aforementioned authors. (Fig. 1.)

At surgery, in the first case the esophagus was normal to inspection and palpation but upon incision and esophagotomy a true diaphragm-like web was found. (Fig. 2.) This was corrected with a local esophagoplastic procedure. The pathologic specimen showed only



1A 1B

FIG. 1. A and B, patients were fifty-five year old men. Severe dysphagia was present in each case. Both patients were operated upon with good results. (From: BUGDEN, W. F. and DELMONICO, J. E., Jr. Lower esophageal web. *J. Thoracic Surg.*, 31: 1–18, 1956.)

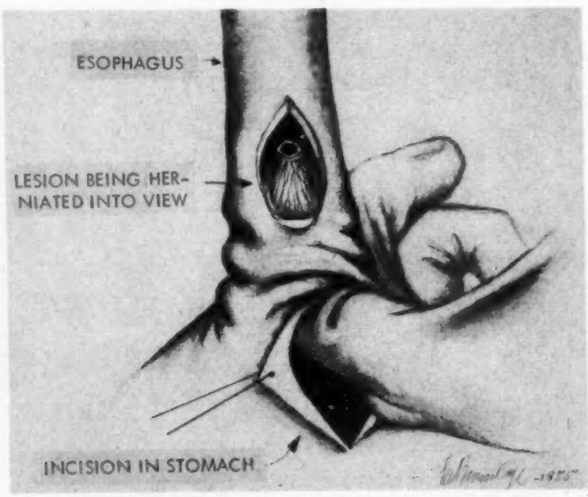
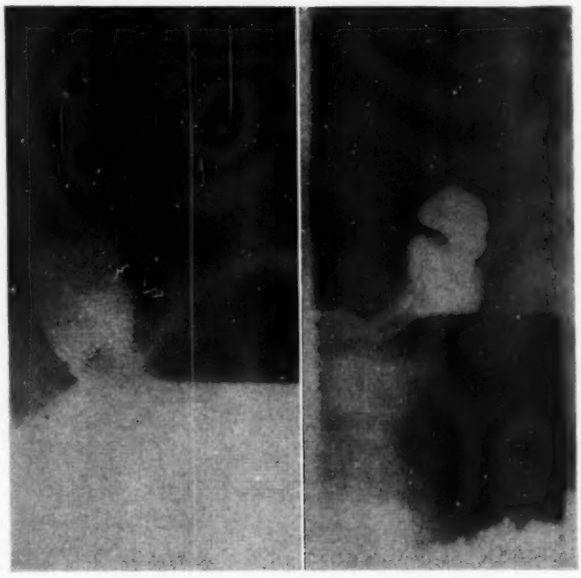


FIG. 2. Schematic drawing of web as seen at operation. (From: BUGDEN, W. F. and DELMONICO, J. E., JR. Lower esophageal web. *J. Thoracic Surg.*, 31: 1–18, 1956.)



3A 3B

FIG. 3. Patient was a twenty-two year old man. A, obstruction caused by piece of steak. B, after removal of foreign body. Excentric deformity, possibly a form of web fractured by esophagoscopy.

hyperplastic squamous epithelium with submucosal inflammatory changes.

The second case was similar historically and radiologically. At operation the lesion was definitely palpable through the intact esophagus. This case differed from Case 1 in that there was a mucosal web and also definite scarring within the muscular wall. At this time the author believes that this was truly a mucosal web but with the submucosal and the muscular scarring of a stricture as well. Unfortunately, a specimen was not taken for microscopic study.

This suggests that "webs" may differ pathologically and that there may be great similarity and/or admixtures between webs and short strictures. On the basis of our present limited knowledge it seems that similar x-ray findings may occur in: (1) lower esophageal web, (2) short strictures, and (3) "contractile esophageal ring" or "lower esophageal ring." Allison of Oxford is of the opinion that webs are of the same morbid anatomy as short strictures—both being due to the inflammatory changes following kissing ulcers—basically caused by the reflux esophagitis due to sliding hiatus hernia.

The pitfalls in diagnosis are twofold. Thin barium will not delineate the lesion. A barium capsule or very thick, pasty barium will readily bring out the web obstruction when previous

FIG. 4. Patient was a fifty year old man with severe dysphagia. Heineke-Mikulicz operation was performed. The condition was probably a short stricture rather than a web. A good result was obtained.

x-ray examinations have been reported as negative. Thin barium will pass through with no hesitation to suggest an obstruction.

Endoscopy may lead to erroneous interpretations. The web may not be visualized as such and the examiner may be uncertain whether the esophagogastric junction is anatomically difficult to pass or whether there is true organic obstruction. The author misdiagnosed the first two reported cases endoscopically.

In a third case there was a sensation of rigid obstruction in the region of the esophagogastric junction but no lesion was visualized. The endoscope suddenly passed over a rigid shelf into the stomach. This forceful maneuver produced some bleeding. (Fig. 3.)

In a fourth case a rigid diaphragm with an excentric lumen approximately 3 mm. in diameter was clearly visualized. (Fig. 4.)

In a fifth case there again was no visualization on endoscopy. The instrument seemed to stop at the 45 cm. level and was then introduced into the stomach blindly over an olive-tipped

FIG. 5. A sixty-eight year old woman with moderately severe dysphagia. There was great improvement after esophagoscopic dilation. The condition was either a web or a short stricture.

bougie. (Fig. 5.) There was a strong sensation of rigid resistance to the endoscope at the level of the obstruction, with sudden release as the obstruction was passed.

SURGERY

The illustrations presented herein indicate the type of plastic procedure employed in the two cases reported by the author. The esophagus is reached through a routine low left posterolateral incision. The web is located through a small longitudinal esophagotomy and then gastrotomy is performed through the incised diaphragm. The left index finger inserted upward will encounter and bring the web into view. (Fig. 6.) Pie-shaped wedges of the web are then resected. (Fig. 7.) Possibly complete circumferential removal will prove to be a better surgical technic. Maintaining a good cardia was thought to be of great importance. If there is an associated sliding hernia as suggested by Allison, concomitant repair of the

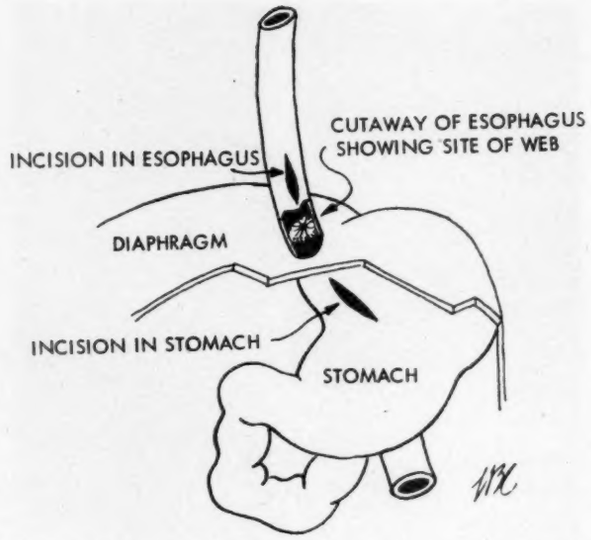


FIG. 6. Schematic diagram of surgical approach. (From: BUGDEN, W. F. and DELMONICO, J. E., JR. Lower esophageal web. *J. Thoracic Surg.*, 31: 1–18, 1956.)

diaphragmatic crura is certainly indicated at operation.

The actual incidence is as yet undetermined, of course, but there have been several personal communications indicating that other thoracic surgeons are now diagnosing and operating upon webs and web-like obstructions at the lower end of the esophagus. More experiences and pathologic studies must be accumulated to substantiate and elucidate the status of this lesion further. As yet the reported experiences of any one thoracic surgeon have been very small in such cases. The amount of pathologic

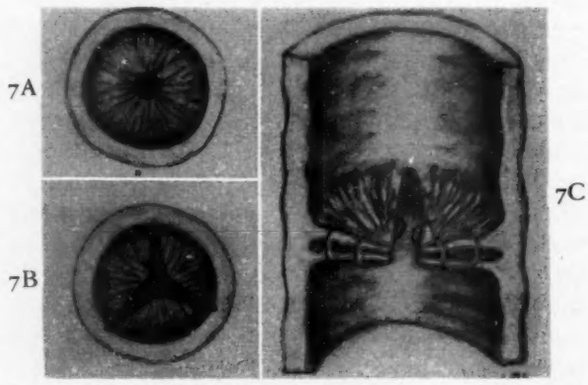


FIG. 7. A, cross section of esophagus from above showing web-like membrane. B, cross section of esophagus from above showing wedge-shaped pieces removed from membrane. C, longitudinal section of esophagus showing sutured flaps of web-like membrane. (From: BUGDEN, W. F. and DELMONICO, J. E., JR. Lower esophageal web. *J. Thoracic Surg.*, 31: 1–18, 1956.)

material available for study has been negligible. It is suggested that surgeons who encounter these lesions in the future take a long, complete longitudinal strip of all layers of the esophageal wall as a specimen for histologic study. The strip should pass through and include a portion of the lesion and could be excised in the course of the repair. This maneuver may not always be feasible, depending on the type of repair employed by the surgeon. In this fashion, however, enough material and opinions will be accumulated to clarify the status of this entity pathologically.

SUMMARY

A new entity causing lower esophageal obstruction, called "lower esophageal web," has been described. Insufficient data regarding its true nature and pathology exist at this time. Similar x-ray findings may occur in (1) lower esophageal web, (2) short stricture or (3) "contractile esophageal ring" or "lower esophageal ring." Endoscopy does not always confirm the differential diagnosis. Severely symptomatic cases deserve exploratory operation and surgical correction.

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Benign Tumors and Cysts of the Esophagus

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BENIGN tumors of the esophagus are among the most gratifying of all esophageal lesions from the standpoint of surgical treatment. Many of these cysts and tumors can be removed without opening the esophageal mucosa and consequently such patients can be operated upon with a low morbidity and mortality.

A number of excellent reviews on this subject have appeared in the literature since that of Adams and Hoover [1] in 1945, when these authors found a total of ninety-four cases and added three cases from this Clinic. In the past ten years a number of isolated cases have been added to the group, including the series of Harrington [2] which contained nine benign esophageal tumors.

In 1951 Myers and Bradshaw [3] reviewed the available literature and found thirty-nine benign tumors in thirty-eight patients. Since this review there have been at least seven further reports. Nevertheless, the total of surgically treated cases reported in the literature is probably still less than 100. When the cases which are presented herein are included, we find a total of only sixty-six cases at the time of this report. In 1954 we [4] reviewed the Lahey Clinic experience and reported fifteen benign tumors appearing up to that time, comprising one polyp, one hemangioma, two fibromas, two cysts and nine leiomyomas. Three of these cases had previously been reported by Lahey [5], by Adams [6] and by Adams and Hoover [1]. Two patients with benign leiomyoma of the esophagus have subsequently been treated.

LEIOMYOMAS

In our experience the leiomyoma is the most common benign tumor of the esophagus. Eleven such cases have been successfully treated.

The first patient was a forty year old man who had complained of dysphagia for three years and had considerable weight loss. On examination he was found to have a pulsion diverticulum of the upper esophagus. Upon excision this diverticulum was found to contain a leiomyoma 2.5 cm. in diameter. The patient had an uneventful convalescence.

This is the only reported instance of association of a benign esophageal tumor with such a diverticulum, although Hoyne and Rogers [7] had previously reported a myoma in association with an epiphrenal diverticulum of the esophagus in a forty-four year old woman. Stewart [8] reported the association of leiomyoma and diverticulum in two patients at postmortem examination.

In 1950 when Goldman and Masters [9] examined the literature on benign tumors of the esophagus they found only seven cases of surgical removal of leiomyomas and added two of their own. The review of Myers and Bradshaw previously cited included twenty-four leiomyomas surgically removed up to May, 1951. At the time of our review in 1954, 112 leiomyomas of the esophagus had been reported, with surgical removal in twenty-six cases and twenty-four survivals. If due allowance is made for unreported cases, the over-all

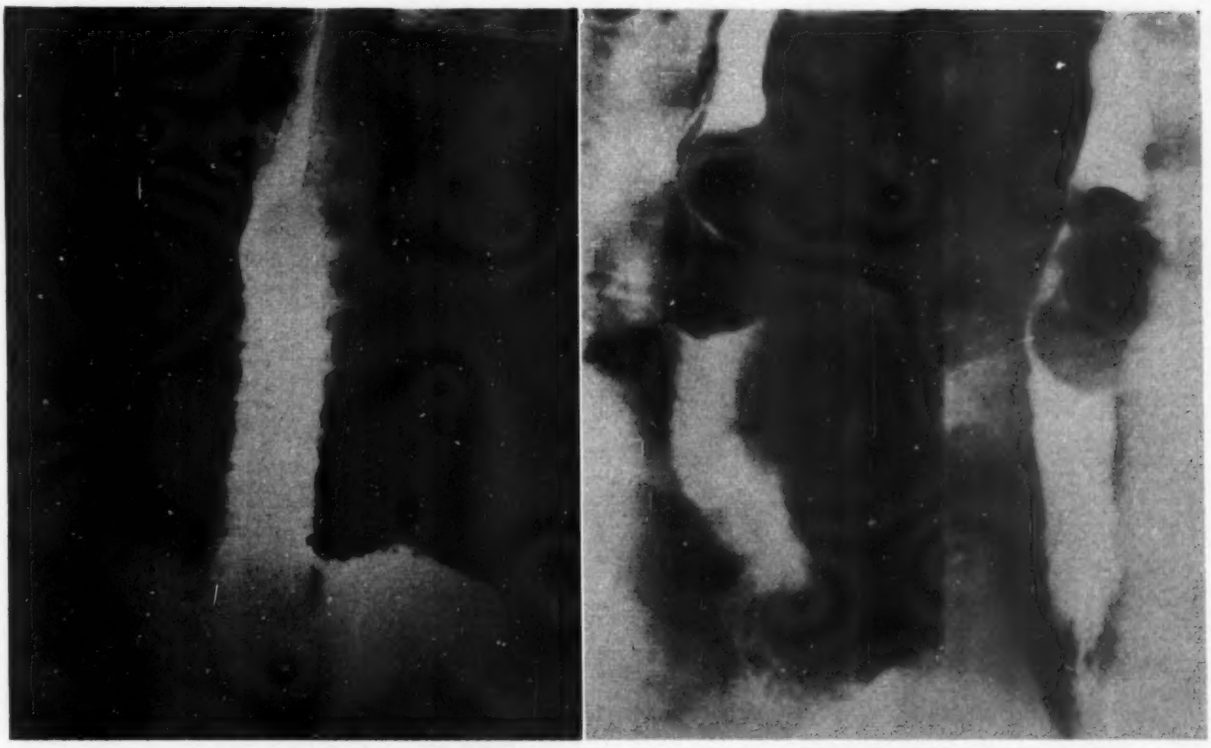

FIG. 1. Typical roentgenologic appearance of a leiomyoma of the esophagus.

incidence of this lesion cannot be large, as shown by the report of Moersch and Harrington [*10*] who found only thirty-two leiomyomas of the esophagus in 7,459 autopsies. Daniel and Williams [*11*] found none in 4,000 autopsies.

The second case in our group proved to be of unusual interest because of the association of pulmonary complications from a massive benign tumor of the esophagus. This patient was a fifty-eight year old woman who had pain in the right lower chest, chills, fever and hemoptysis of six months' duration. Roentgenologic examination revealed a large mass in the right hemithorax displacing the esophagus anteriorly and producing atelectasis of the right lower lobe. At operation the lesion was shown to be a leiomyoma weighing 148 gm. and measuring 10 by 7.5 cm., attached to the lower third of the esophagus. A right middle and lower lobectomy was necessary in addition to excision of the mass. The esophageal lumen was not entered during the surgical procedure and the patient made an uneventful recovery.

Examination of the records of eleven cases of leiomyoma of the esophagus reveals that six of these had no symptoms referable to the esophagus. In these cases the lesions were discovered at routine examination. (Fig. 1.) The remaining five patients had dysphagia, epigastric burning and substernal pain. In these eleven cases the lesions were distributed as follows: four were in the lower fourth of the esophagus, four in the middle half and three in the upper fourth. In all but the first of these cases the leiomyomas were removed without entering the mucosa of the esophagus. All patients made an uneventful recovery and none has had recurrence of symptoms.

CYSTS

The complex nomenclature associated with intrathoracic cysts of gastrointestinal origin attests to the difficulty in determining their origin. Thus they have been called intrathoracic cysts of foregut origin, periesophageal cysts, diverticula of the esophagus, duplications of the alimentary tract, conglomerate gastroenteric cysts, gastrogenic cysts, accessory esophagus and accessory stomach. Cysts that are not attached to the esophagus are usually classified as mediastinal cysts although they may actually be esophagenic or bronchogenic in derivation.

The first report we have been able to find of a mediastinal cyst lined with gastrointestinal mucosa is that of von Wyss [*12*] in 1870. Since

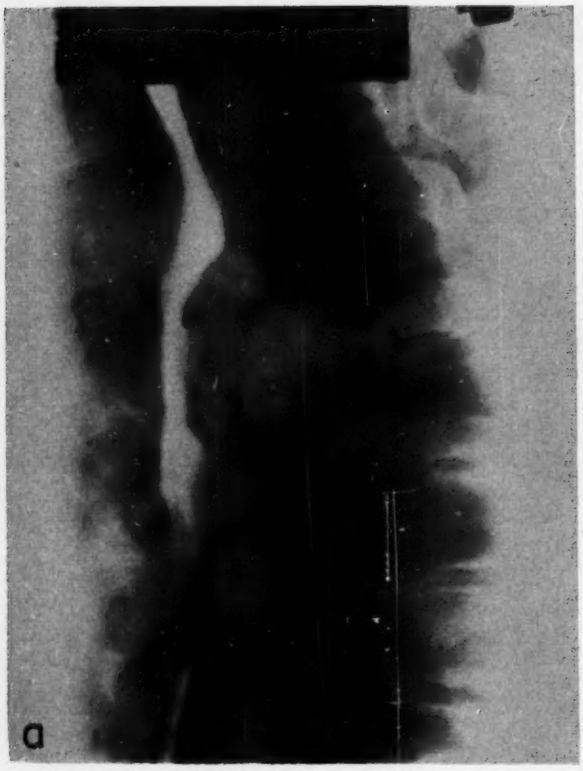

FIG. 2(*a*). Barium study of esophagus showing filling defect and soft tissue mass. Lesion proved to be an intramural esophagenic cyst.

FIG. 2(*b*). Photomicrograph of cyst shown in (*a*). Cyst is lined with ciliated columnar epithelium and its wall is covered with smooth muscle; no cartilage was present. (From: BOYD, D. P. Lesions of the superior mediastinum: diagnosis and treatment. *S. Clin. North America*, 33: 835, 1953.)

then a number of reports of mediastinal enteric cysts have appeared. Some of these cysts have been related to the esophagus and some have not.

In 1946 Olenik and Tandatnick [*13*] collected seventy-four cases and added a case of their own, while Lindquist and Wulff [*14*] in 1947 found eighty cases, including the aforementioned group.

Myers and Bradshaw found eleven cases in the literature in which surgical removal had been attempted for esophagenic cysts. Five cases not included in this review had previously been reported in 1944 by Ladd and Scott [*15*]. Sabiston and Scott [*16*] reported two additional cases in 1952, one an autopsy report and the other an unsuccessful attempt at excision in a two year old child.

Two cases of esophagenic cysts have been found in the Lahey Clinic experience and thus a total of approximately 104 mediastinal cysts of enteric origin have been reported. In only thirty-nine of these, however, was it specifically stated that they were related to the esophagus. Surgical removal was attempted in seventeen cases and fourteen patients survived. Sauerbruch [*17*] is believed to have performed the first successful excision of an esophageal cyst in 1937.

The first patient, a forty-eight year old man, had no esophageal symptoms. A posterior mediastinal mass with compression and displacement of the esophagus was found during a routine examination. The lesion protruded slightly into the right hemithorax. (Fig. *2a* and *b*.) The cyst was located beneath the arch of the azygos vein at thoracotomy and was covered by the longitudinal muscle fibers of the esophagus. Separation from the esophageal mucosa was difficult but it was possible to remove the tumor without opening the mucosa. The specimen was a smooth-walled, unilocular cyst measuring 6 by 7 by 6 cm., containing thin, pearly gray fluid. Microscopic examination revealed the cyst wall to be lined with ciliated, pseudostratified, columnar epithelium with a well defined submucosa and a thick muscularis.

The second cystic lesion was in a forty-seven year old man who also had no esophageal symptoms. Roentgenologic study after a barium swallow revealed the characteristic smooth indentation of the esophageal wall typical of benign tumor. At operation a cystic mass was found in the left lateral wall of the esophagus near its lower end; it measured 4.5 by 2.5 cm. The esophageal mucosa was not opened during removal. The contents of the cyst proved to be inspissated mucus. The muscular wall of the esophagus was readily approximated with interrupted stitches. Microscopic study of the cyst showed it to be lined with orderly ciliated, pseudostratified, columnar epithelium.

Considerable speculation has centered around the origin of esophagenic cysts. Small diverticula of the foregut occurring in early fetal life may be pinched off and epithelial nodules misplaced, with subsequent cystic change. This is the theory of Lewis and Thyng [*18*]. Olenik and Tandatnick have modified this theory and suggest that all enteric cysts of the mediastinum are derived from the foregut at or near the origin of the lung bud. In this congested area the primordial cells of lung and esophagus develop very close together. Pinching off of single cells or small groups may form the nidus of a later cyst. The work of Johnson [*19*] and of Forssner [*20*], later elaborated upon by Lewis [*21*], showed that the esophageal wall at the 10 mm. stage contains large folds and vacuoles. Bremer [*22*], Keith [*23*], and Keibel and Mall [*24*] have suggested that failure of these vacuoles to coalesce with the lumen of the esophagus brings about a persistence of this space which gives rise to an esophagenic cyst. This hypothesis of the origin of congenital cysts of the esophagus appears to be the most reasonable one at this time.

The wall of a typical esophagenic cyst contains smooth muscle fibers with a moderate amount of loose areolar tissue. The lining may be ciliated. It is to be emphasized that the presence of cilia does not necessarily indicate respiratory origin. Neumann [*25*] in 1876 showed that in the eighteen- to thirty-two-week embryo the esophageal lining contains areas of non-ciliated and ciliated epithelium. This observation was confirmed by Keith. Furthermore, ciliated epithelium may be found lining the esophagus of the newborn. A reasonable explanation is available, therefore, for the fact that an intramural cyst of the esophagus may be lined with gastric, enteric or respiratory epithelium.

From a practical standpoint, esophageal cysts are relatively harmless. This is in marked contradistinction to mediastinal cysts of gastric origin which may enlarge rapidly, may ulcerate or give rise to respiratory symptoms or hematemesis.

FIBROMAS

Chi and Adams [*26*] surveyed the literature and found only nine cases of fibroma of the esophagus up to 1948. These authors added a case of excision of a fibroma performed by W. E. Adams in 1942 and suggested that this might be the first successful removal. However, Lahey had previously removed a fibroma of the esophagus on March 7, 1941, with a successful outcome. Two fibromas which are included in the Lahey Clinic series were apparently the first to be removed successfully. The rarity of this lesion is shown from the review of Moersch and Harrington and that of Schafer and Kittle [*27*]; in 13,500 autopsies no fibromas were found.

A forty-eight year old woman reported at the Clinic complaining of shortness of breath. Fluoroscopy and barium studies revealed a soft tissue mass low in the cervical region displacing the esophagus anteriorly. The mass moved with swallowing. The preoperative diagnosis was intrathoracic goiter. At operation the mass was, therefore, exposed through the usual collar incision. Upon exposure, however, it was seen to be lying posterior to the recurrent laryngeal nerve and the inferior thyroid artery. This site suggested that the lesion was not a goiter, since in none of the many thousands of patients with goiter treated at this Clinic has the recurrent laryngeal nerve been found to lie anterior to the gland. The tumor was enucleated from the esophagus without difficulty. The patient made a smooth convalescence. Microscopic study of the specimen showed the lesion to be a fibroma.

A forty-eight year old woman first came to the Clinic in 1942 and stated that for many years she had had difficulty in swallowing. A few weeks before admission she had regurgitated food and on one occasion hemorrhage had occurred. Esophagoscopy revealed a large, globular, smooth-surfaced mass in the upper part of the esophagus attached to the right

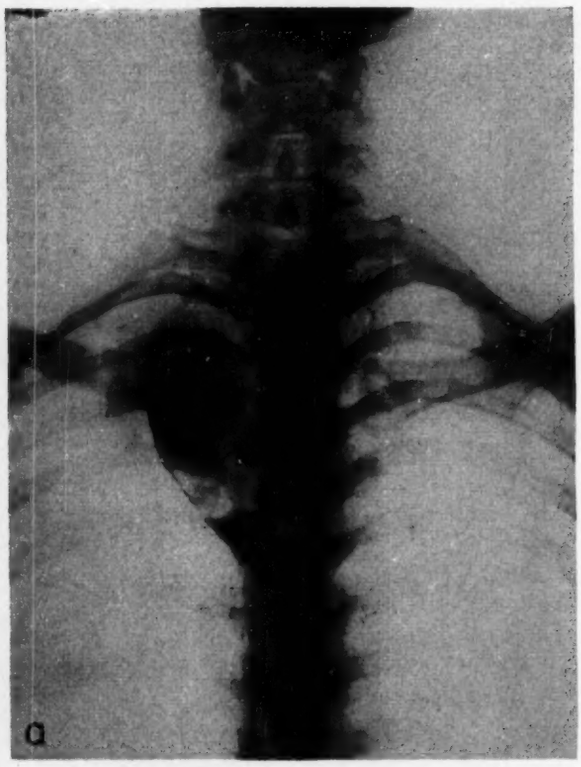

FIG. 3(*a*). Fibroma of the esophagus.

FIG.3 (*b*). Photomicrograph of fibroma shown in (*a*).

posterolateral wall. There was almost complete obstruction, and superficial ulceration of the mucosa was noted. The dangers of mediastinitis and empyema were much greater at that time than they are today, so preliminary gastrostomy was performed followed with thoracotomy. The lesion was removed without difficulty and the defect in the mucosa was sutured. The patient was fed through the gastrostomy tube for two weeks. She made an uneventful recovery. Microscopic examination revealed the mass to be a fibroma. (Fig. 3*a* and *b*.)

POLYPS

Polyps of the esophagus are of special interest because they were among the first lesions of the esophagus to be treated surgically. Monro in 1763 ligated the pedicle of an esophageal polyp, following which the tumor was passed in the stool. Dubois [*28*] in 1818 was less fortunate when he employed the same maneuver. The tumor separated and was aspirated into the trachea, and the patient died. Since then the treatment of choice in these lesions has been esophagoscopy and snare removal. The patient included in this series came to the clinic complaining of dysphagia of some years' duration. Several days before admission he had become partially asphyxiated and regurgitated a polyp into the back of his throat. He was finally able to swallow the tumor and push it back into the esophagus manually. Esophagoscopy revealed a polyp of the esophagus and recent hemorrhage. On December 2, 1942, using an endotracheal tube to secure an adequate airway, the lesion was delivered into the mouth. It was then removed with a snare. Follow-up study indicated that the patient had no further symptoms. The pathologic report was polyp of the esophagus. (Fig. 4.)

HEMANGIOMAS

Hemangioma of the esophagus, fortunately, is rare. In the two large autopsy series previously referred to, no hemangiomas were found. However, in their comprehensive review of vascular lesions of the gastrointestinal tract in 1949, Gentry, Dockerty and Clagett [*29*] found sixteen benign vascular tumors of the esophagus, one of which may have represented varices. Twelve were autopsy findings and in four patients the tumors were recognized

clinically. Of these four, three were treated and one died of hemorrhage without treatment. It is significant that all three patients with symptoms from hemangiomas were dead within one year after onset in spite of treatment. In 1926 Vinson, Moore and Bowing [30] attempted to treat a benign vascular tumor of the esophagus with radium. The patient died twelve months later from esophageal obstruction. Palanker, Constantine and Paine [31] reported successful resection of a hemangioma of the esophagus in 1951.

A forty-four year old woman came to the clinic because of recurring hematemesis over a twenty-year period. She had had five episodes in all, the last occurring one year before admission. Barium studies revealed that the esophagus was dilated and tortuous. There were numerous filling defects in the upper two-thirds. Complete studies showed no evidence of vascular or hepatic disease. Esophagoscopy on November 8, 1941, revealed large blue folds protruding into the lumen of the esophagus and involving the upper two-thirds; the lower third was reasonably normal. No treatment was carried out. This patient has been followed up for fourteen years; she is well and free from esophageal bleeding at this time.

Despite the favorable result in this case, it is apparent from a review of the literature that hemangioma of the esophagus is an exceedingly dangerous lesion and, if localized, should probably be treated aggressively.

COMMENT

In only seven of the seventeen cases reported herein were complaints specific with regard to the esophagus. The diagnosis was determined accurately in thirteen of the seventeen cases. In one case, as already noted, a fibroma was confused with an intrathoracic goiter and in another case a benign leiomyoma was thought to be a pericardial cyst. In another case, a leiomyoma lying posteriorly close to the paravertebral gutter was thought to be a neurogenic tumor of the posterior mediastinum. Careful roentgenologic study, however, will indicate benign esophageal lesions with a high degree of accuracy. Reference is made to the excellent paper of Schatzki and Hawes [32] in this regard.

Since the diagnosis can usually be made preoperatively, biopsy of the esophagus is unnecessary and for obvious reasons undesirable.

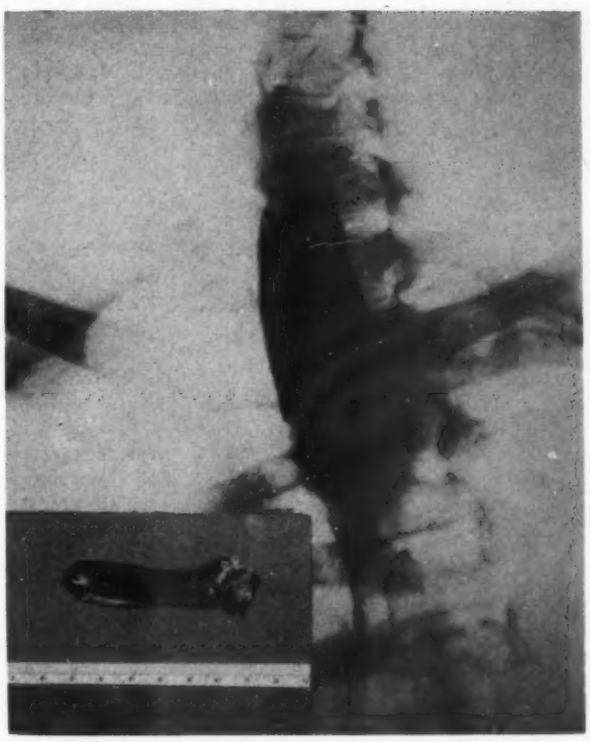

FIG. 4. Esophageal polyp. Insert, excised specimen.

SUMMARY

Seventeen cases of benign tumor of the esophagus are presented, with sixteen successful surgical removals. There were no deaths, no hospital morbidity and no recurrence of symptoms.

The literature relative to this subject has been briefly reviewed.

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Carcinoma of the Esophagus

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CANCER of the esophagus has engaged the serious attention of many capable surgeons, pathologists, endoscopists and internists during the past half century. The radiation therapist is concerned with this disease and has added to the widespread interest of the medical profession, and considerable literature on this subject has accumulated.

Much original thought, experimentation and imagination has gone into the development of the many and varied forms of treatment which have been advocated at one time or another for the cure of cancer of the esophagus. Noteworthy among these methods was the advent of radium as an intraesophageal method of radiation. This form of treatment was first proposed in 1904 and soon afterward the literature was flooded with descriptions of many new types of apparatus, new dosages, new filters and new methods for accurately locating the radium in the esophagus. This period which began so hopefully was not, however, followed by reports of cures or even good palliative results and the method soon fell into disfavor.

A great deal of attention has been directed toward the development of a procedure for the radical, surgical removal of carcinoma of the esophagus. History records that the first successful, intrathoracic esophagectomy was accomplished in this country by Torek in 1913 [7]. With this demonstration that the esophagus could successfully be removed and that a patient with cancer of that organ could be cured, it was natural to suppose that the management of esophageal cancer by means of surgery would be just a matter of technical skill, and surgery was thought to have reached a point where it could be called an answer to the problem of esophageal cancer. Unfortunately, surgery has not fulfilled its early promise. Such a general lack of success would lead one to suspect that those who have advocated the various methods of treatment have been unjustifiably optimistic.

A careful study of the clinical course of the disease, the anatomy of the organ and the postmortem material available will show that when cancer of the esophagus is first diagnosed the patients are usually in a stage in which their disease is not curable by any of the methods of treatment available at the present time. Therefore, in most cases of esophageal cancer the treatment of choice should be directed toward palliative management.

The material for this report has been taken from the records of Memorial Hospital accumulated during two periods of our experience. The first series was accumulated between 1918 and 1931. Five hundred six patients with carcinoma of the esophagus were admitted to the hospital during that interval.

The second group of patients was admitted to Memorial Hospital between 1931 and 1955; a total of 1,250 patients were admitted during this interval. Therefore, the total number of patients with cancer of the esophagus suitable for study is 1,756. The original group of 506 patients has previously been reported [11]. It must be noted that this material consists of all patients who come to Memorial Hospital with cancer of the esophagus regardless of the stage of their disease. Almost all of our patients are in the advanced stage of the disease; a high percentage are in a hopelessly advanced stage and are even moribund at the time of admission. Many of the patients have undergone surgical and radiation therapy at other institutions before coming to us. The high mortality rate and the low salvage rate for this disease is well known, and when the material is so heavily weighted with hopeless cases the study of it naturally paints a very gloomy picture.

ETIOLOGY

It is, of course, impossible to give exact etiologic data regarding cancer of the esopha-

gus. However, it is now quite generally believed that there are certain definite factors predisposing to esophageal cancer. In the oral cavity it has been shown that broken, irregular or sharply worn teeth, ill-fitting dentures, leukoplakia, excessive pipe smoking, syphilis and

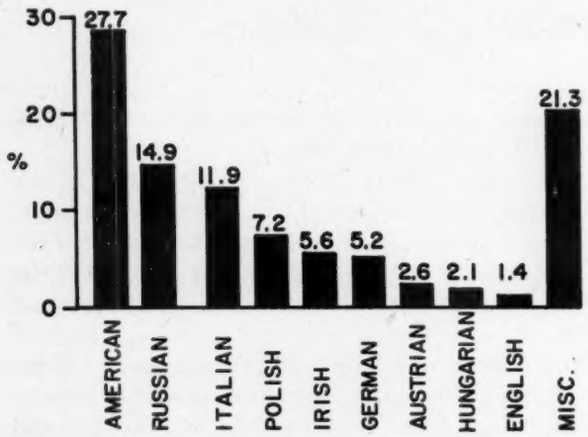

FIG. 1. Racial distribution of cases in percentages.

intraoral sepsis are definitely irritating factors and in some cases at least exert a carcinogenic action in the causation of intraoral cancer.

Similar factors prepare the esophageal mucous membrane for precancerous changes. Badly kept teeth, intraoral sepsis and ill-fitting plates tend to cause hasty and incomplete mastication which makes it necessary for the esophagus to handle masses of food which are often too large and too hard. Such a large, hard food bolus causes a certain amount of trauma and inflammation of the esophageal mucous membrane. The passage downward of such a bolus would be slowest at the points of anatomic constriction and resulting trauma is greatest, therefore, at these points. The trauma causes irritation which leads to spasm and with it further slowing of the food bolus and later even stagnation, esophagitis, ulceration and possibly neoplasm.

Thermal irritation is probably the most constant factor predisposing to carcinoma of the esophagus. The frequent drinking of copious amounts of excessively hot tea is a history frequently obtained from the Russian patient with cancer of the esophagus. This fact is especially significant when it is noted that 46 per cent of the foreign-born patients in our first series were born in Russia. There were more Russians than native Americans. In China, cancer of the esophagus is very uncommon among the women and this is said to be due to the fact that they eat after the men have finished and their rice is not as hot. The women of Scotland drink excessively hot tea and Turner [8] has reported that cancer of the esophagus is more common in them than it is among the men. (Fig. 1.)

Leukoplakia of the esophagus is being recognized more frequently due to the increasing popularity of the esophagoscope. We have found it to be a common condition and it has been reported to have been present in 60 per cent of 200 non-cancerous cases autopsied. Clinically and pathologically, the process is the same as that seen in the oral cavity where it is definitely a precancerous condition [6].

Others who have had extensive experience with esophageal cancer think that the excessive use of alcohol, spiced food and tobacco are factors in many cases. Others have reported that cancer of the esophagus is often definitely attributed by the patient to some particular mental or physical shock. It has been observed that there was an increase in the incidence of cancer of the esophagus in young adults following World War I. It was thought that the following sequence of events had taken place: first an emotional upset, then esophageal spasm and finally stasis leading to esophagitis stricture and finally cancer. Jackson [3] says that cancer of the esophagus occurs in patients who have had esophageal spasm for years. Mosher [5] demonstrated the presence of thin webs and pouches in the esophagus and these were thought to have some etiologic bearing as the cicatricial contractures which result from the swallowing of caustic solutions or result from the healing of peptic or traumatic ulcers. In one of the youngest male patients in our series, cancer of the esophagus developed in a cicatricial scar resulting from the ingestion of lye when he was three years old. He was twenty-eight when cancer of the esophagus appeared.

Syphilis does not play an important role in the etiology of esophageal cancer and in our series it was present in about the same proportion as it is in the general population.

INCIDENCE

Slightly more than 2.5 per cent of all the patients admitted to Memorial Hospital with malignant disease have cancer of the esophagus. In New York City, where more than 8,000 cancer deaths occur each year, it has

been shown that about 280 of these are due to cancer of the esophagus, an incidence of about 3.5 per cent. In England cancer of the esophagus is said to comprise about 5 per cent of all cancer deaths, and others have reported an incidence of as high as 10 per cent.

SEX

It is well known that this disease is much more common in the male than in the female. Guisez [2], in reporting 1,430 cases, found the ratio to be six males to one female. Jackson [5] with 671 cases, reported an 87 per cent incidence in males. In our second series of 1,250 patients, there were 1,011 males and 239 females, or a ratio of four males to one female.

AGE

The greatest number of patients, both male and female, were between the ages of sixty and sixty-nine. In eleven of twelve patients cancer developed between the ages of fifty and seventy-nine. It is interesting to note that we have had twenty-two male patients and two female patients in whom cancer of the esophagus developed after the age of eighty. Our figures also seem to indicate that in women cancer of the esophagus develops at a slightly earlier age than in males. (Fig. 2.)

SYMPTOMATOLOGY

The onset of carcinoma of the esophagus is a most insidious one and although we have a ready means (esophagoscopy) for making a positive diagnosis, it is still a fact that one seldom sees a patient in the early stages of esophageal cancer. The function of the esophagus is to transport food from the pharynx to the stomach and this function is only seriously interfered with late in the course of the disease. Then, too, the patient tends to masticate his food more thoroughly after he first notices that large particles of food have a tendency to stop or stick part way down the gullet. Such periods of temporary dysphagia tell a story to the physician, but unfortunately the patient pays little heed to such a matter and will mention it only when a careful history is obtained.

When definite, persistent dysphagia occurs and the patient seeks relief for it, his disease is usually well advanced. It is most unfortunate that dysphagia should so often be the first symptom of esophageal cancer. In our series almost two-thirds of the patients gave as their first symptom difficulty in swallowing solid foods. If the mortality from this disease is to be reduced, earlier diagnoses must be made and earlier symptoms must be sought for and detected. When dysphagia, dehydration and emaciation exist, the disease is almost always hopelessly advanced and incurable. Dysphagia alone is not an infallible diagnostic symptom of esophageal cancer as it may occasionally be entirely absent even in patients dying of the disease, and in other cases it may have been present for twenty years or more as a result of so-called cardiospasm. Dysphagia is thus an unreliable symptom and of little aid in making an early diagnosis.

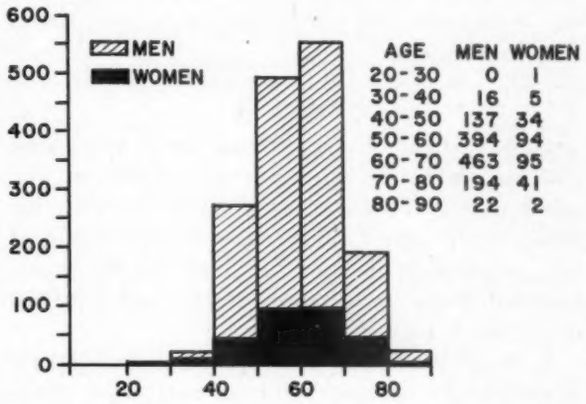

FIG. 2. Distribution of ages of patients admitted for treatment, men and women compared.

Under early symptoms we have occasionally noted vague complaints such as a feeling of substernal pressure, a feeling of obstruction, hiccoughs, hoarseness, difficulty in breathing, heartburn and increased mucus. Foul breath is a common symptom.

Pain is not an early symptom and when backache, substernal discomfort or pain do exist it follows that the disease has extended beyond the esophageal walls into the posterior mediastinum. Hoarseness, when present, is usually due to involvement of the recurrent laryngeal nerve and is most commonly noted on the left side. Regurgitation, vomiting, loss of weight, weakness and pain are all symptoms of a late stage of the disease. In making our plea for earlier diagnosis it is well to recognize that the early symptomatology is often too vague to be of much help in this direction. We undertook a study of well patients in which we took an esophagram of every person over the

age of forty, hoping in this way to discover early cases of esophageal cancer. A very large number of patients were studied in this manner and only one tumor of the esophagus was discovered; although this patient's growth was resectable and she has remained well for eleven years, the investigation was considered to be too expensive, too time-consuming and therefore impractical.

DIAGNOSIS

Cabot [9], in his report on 3,000 autopsies, showed that the diagnosis of cancer of the esophagus could be completely missed. In his series there were twenty cases of carcinoma of the esophagus; in four of these the diagnosis had not been made before death and in three it had been merely suspected.

It must be emphasized that esophagoscopy is absolutely necessary for an early and accurate diagnosis of esophageal cancer. Complicated apparatus, difficult x-ray technics and various opaque mixtures have been developed so that an early roentgenographic diagnosis is possible, but these have lost their popularity to some extent due to the more satisfactory esophagoscopic results.

The following diagnostic procedures are carried out at Memorial Hospital, during the investigation of a patient suspected of having carcinoma of the esophagus. Following the usual history and general physical examination the patient is given a more detailed examination of the head and neck, oral cavity, larynx and the like, and blood is taken for a Wassermann test and liver studies. Following this he is sent for fluoroscopy and barium swallowing, and then x-ray films are taken of the esophagus and lungs. The following day the patient is taken to the endoscopic room; the pharynx is cocainized, esophagoscopy is performed and a biopsy specimen obtained.

Fluoroscopy is essential for a complete examination because by this means we are able to observe the function of the esophagus. The procedure also determines the following points regarding cancer in this organ: size, shape, outline and position of lesion, as well as the size and shape of the lumen above and below the obstruction. This information is of the utmost value in determining what form of treatment should be instituted. We believe that the endoscopic procedures should be performed by the surgeon who is to direct the future therapeutic management of the patient whether this be by surgery or radiation. At the completion of the esophagoscopy the trachea and main bronchial tree is examined through the bronchoscope for evidence of possible direct extension of cancer from the esophagus.

TREATMENT

The majority of our patients coming to Memorial Hospital with cancer of the esophagus are in the advanced stage of the disease and the question of surgical management of the cancer cannot seriously be considered. Radiation alone or in conjunction with supportive surgical measures such as gastrostomy has therefore been called upon to shoulder most of the responsibility for the palliative management of these patients.

External radiation, whether given by the conventional 250 k.v., the 1 m.e.v., or the 60 cobalt units, requires for best results a very careful and proper outlining of the treatment portals in order to cross fire the beam of radiation accurately in the thoracic cavity at the level of the cancer. These portals must be mapped out on the skin of the chest anteriorly and posteriorly under fluoroscopic control with the patient in the position in which he is to receive treatment. The upper and lower limits of the cancer are marked with lead strips both posteriorly and anteriorly.

It is well known that cancer of the esophagus spreads longitudinally by way of the submucosal lymphatics, and for this reason an area of tissue much greater than that shown on the roentgenograph or by endoscopic study must be included in the beam of radiation in order to obtain a satisfactory result. Skin portals smaller than 14 by 7 cm. are therefore seldom used. We commonly employ four portals, two anterior and two posterior, leaving a free zone of 4 cm. in the midline anteriorly and posteriorly to preserve that portion of skin from possible damage due to cross firing of the beam of radiation.

We have employed intraesophageal radium therapy in a group of patients who have had preliminary gastrostomy. A 30 mg. radium element tandem applicator with a total filtration equivalent to 2 mm. of brass has been found to have a most beneficial effect in reducing tumor infection, healing ulceration and widening the lumen of the esophagus at the tumor site. In our hands it has, however, failed

to eradicate the cancer completely in any individual case.

Radon in the form of gold-filtered seeds has been used frequently. The seeds are inserted at periodic intervals by means of the esophagoscope. For the most part, these gold-filtered radon seeds in adequate dosage have a remarkable tendency to melt down the cancer within a zone of 1 cm. of their site of application, but here again the results have not been satisfactory because intensive radiation produces excessive tumor sloughing and this in turn is often followed by perforation of the esophagus and fatal mediastinitis. An additional technical difficulty is the accurate placement of the gold-filtered radon seeds in the distal portion of the tumor through the esophagoscope. (Table 1.)

SURGERY FOR CANCER OF THE ESOPHAGUS

Most surgeons agree that a satisfactory discussion of surgical measures for the treatment of cancer of the esophagus should be divided into three parts based on the anatomy of the organ under consideration. It seems quite clear that the cervical esophagus, the mid-thoracic esophagus and the abdominal portion of the esophagus vary so greatly in their structure, location and curability that they need to be discussed as separate surgical problems.

Cancer of the Cervical Esophagus. We have had 115 such cases and there have been seventy-five operations. It seems clear that eradication of the cancer in the cervical esophagus requires radical measures and our patients are therefore treated by combining a unilateral, radical neck dissection with complete thyroidectomy and laryngocervical esophagectomy. It has also been found that removal of the first portion of the sternum makes for an easier excision of the neck tissues and at the same time permits a limited dissection of the superior mediastinum. Nineteen of these patients who underwent excisional surgery have survived long enough to permit reconstruction of the esophagus by plastic procedures [9]. An additional four patients with cancer of the cervical esophagus underwent total esophagectomy with anastomosis in the neck performed between the stomach and the hypopharynx.

Cancer of the Mid-thoracic Esophagus. From the surgical point of view it is obvious that cancer of the thoracic esophagus will prove to be difficult to manage and the salvage rate will be low. This is because the thoracic esophagus is deeply situated and intimately surrounded by important and vital structures which are often invaded early in the natural course of the disease.

In the thoracic esophagus ulcerating, badly infected cancers are apt to develop which metastasize readily by way of the rich submucosal lymph vessels into the adjacent mediastinal lymph nodes. It is important to note that these same intramural lymphatic vessels also distribute the neoplastic cells in a longitudinal direction, often to a considerable distance above and below the visible and palpable limits of the primary cancer. Viable tumor

TABLE 1
METHOD OF TREATMENT IN CASES OF CANCER OF ESOPHAGUS (1918–1955)

Treatment	No. of Cases	Average Length of Life after Admission (mo.)
External radiation only:		
Moderate	239	6.2
Inadequate	30	3.3
Intraesophageal radiation only:		
Radium in capsules	12	4.
Radon seeds	3	5.75
External plus intraesophageal radiation:		
Radium in capsules	5	4.3
Radium in capsules and radon seeds	1	6.
Radon seeds	5	10.8
Gastrostomy only*	139	3.5
Esophagogastrostomy (by-pass) only	3	1.5
Gastrostomy plus external radiation	404	7.6
Esophagogastrostomy (by-pass) plus external radiation	9	11.0
Gastrostomy plus intraesophageal radiation:		
Radium in capsules	24	4.3
Radon seeds	3	8.5
Gastrostomy plus external and intraesophageal radiation:		
Radium in capsules	27	5.7
Radium in capsules and radon seeds	3	10.2
Radon seeds	17	18.8
Resection only	126	11.3
Resection and external radiation	56	20.5
No treatment	95	3.1

* Patients treated only by gastrostomy were those whose general condition was too poor to permit palliative radiation.

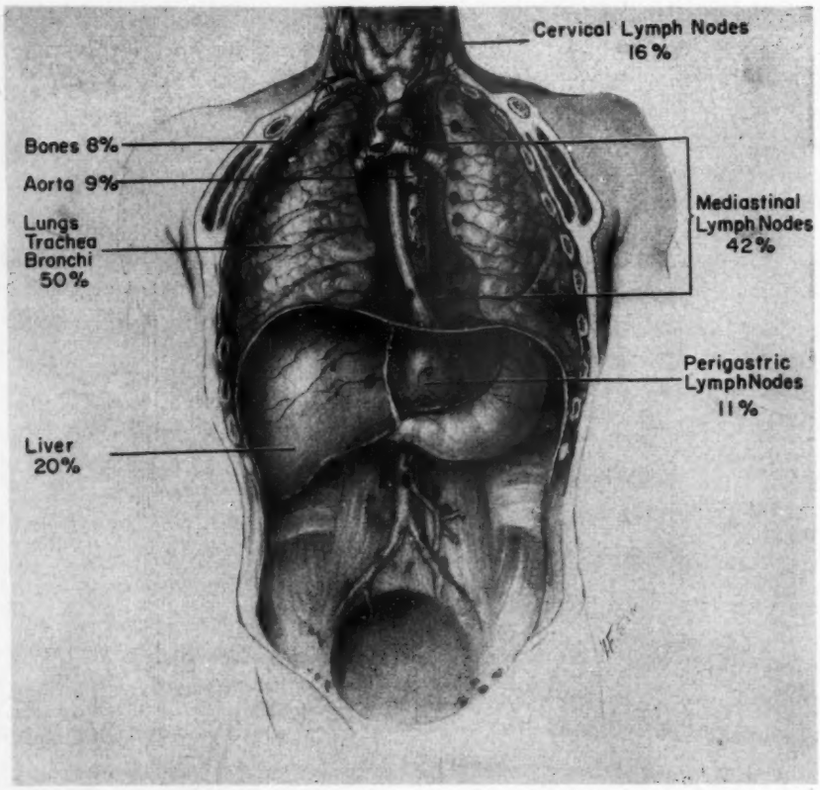

FIG. 3. Diagrammatic sketch illustrating the relative frequency with which the various organs and lymph node areas are involved in secondary deposits from carcinoma of the esophagus. The diagram is based on 185 postmortem examinations.

emboli may block a lymphatic branch and give rise to a secondary deposit of cancer appearing as a submucosal outcropping sometimes as much as 8 cm. distant from the primary cancer. (Fig. 3.)

An early review of our cases of esophageal resection indicated a higher incidence of inadequate longitudinal excision than we had anticipated. Seventy-nine cases were analyzed and 45 per cent of the patients with esophageal cancer who were subjected to segmental resection had an inadequate longitudinal removal of their disease. Surgical failure was established on a critical microscopic basis.

TABLE II

END RESULTS IN CASES OF ESOPHAGEAL CANCER
(1,250 CASES, 1931–1951)
FIVE-YEAR SURVIVAL AFTER TREATMENT

Treatment	No. of Cases	Five-Year Survival	Percentage	Location of Cancer
Surgery alone.....	77	3	4	Cervical esophagus, 1 Distal esophagus, 2
Radiation alone....	929	6	0.65	Cervical esophagus, 3 Upper thoracic, 1 Mid-thoracic, 1 Lower esophagus, 1
Combination of surgery and radiation	39	3	7.7	Cervical esophagus, 2 Mid-thoracic, 1
Total five-year survivals.....	1,045*	12	0.96	

*Two hundred five cases lost to follow-up.

In the hope of cutting down the local recurrence rate and possibly increasing the cure rate, it was apparent in dealing with intrathoracic esophageal cancer that a longer portion of the organ needed to be removed. Therefore, it was decided to perform a series of Torek type esophagectomies. Forty-eight total esophagectomies for cancer were performed; thirty-one of our forty-eight patients who underwent total esophagectomy for cancer survived long enough to permit esophagoplasty.

In the last seven total esophagoplasties we have employed right colon retrosternal transplants. This operation is not a new idea as it was first reported in 1911 by Kelling [4]. The surgical technic and our results have been

published [*10*]. It is our present opinion that when the thoracic esophagus is the site of cancer, complete esophagectomy is indicated whenever feasible and the most satisfactory method for reconstruction of the intestinal tract is a right colon substernal complete esophagoplasty.

Cancer of the Cardiac and Abdominal Portion of the Esophagus. In this region the prognosis is better than for any other segment of the esophagus. Here too the surgeon is permitted to perform a more radical operation with lymph node dissection, and the immediate reconstruction of the excised segment of esophagus can readily be accomplished using the stomach. (Table II.)

END RESULTS IN ESOPHAGEAL CANCER

In a series of 1,250 cases, including "all comers" regardless of condition of patient and stage of disease, there have been only twelve survivals for five years or longer. Seventy-seven patients were treated by surgery alone and there have been three survivals, a 4 per cent five-year survival rate.

Nine hundred twenty-nine patients received only radiation and six have survived more than five years for a salvage rate of 0.65 per cent.

Thirty-nine patients received both surgery and radiation treatment, and there were three survivals, or 7.7 per cent.

In six patients (50 per cent) in the group of five-year survivals, the cancer was located in the cervical esophagus. The prognosis would appear to be more favorable in this anatomic area.

It should be noted that patients with cancer of the stomach, secondarily invading the terminal esophagus, have not been included in our statistics. Such patients, when managed surgically, have a fair prognosis with an improved salvage rate. For these reasons a surgeon may be tempted to include them with other cases of esophageal cancer and so improve his over-all five-year survival figures.

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Endoscopic Intubation and Dilatation in the Management of Esophageal Carcinoma

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IN spite of significant recent advances in the management of carcinoma of the esophagus, many patients are still seen in whom surgery is impossible even for palliation. Some patients in this group may obtain relief from obstructive symptoms by non-surgical methods. Radiation therapy, although useful, does not immediately relieve total esophageal obstruction with its associated aspiration pneumonitis and cough.

Souttar described endoscopic intubation for obstructing esophageal carcinoma before present day excisional technics were developed [1]. Benedict emphasized the usefulness of esophageal dilatation in malignant disease [2]. These methods, used individually or in combination, will allow reasonably satisfactory nutrition in the majority of "inoperable" patients. Using this approach, we have reduced the number of explorations for inoperable cancer and have totally abandoned the distressing and unsatisfactory gastrostomy. We have not performed transthoracic insertion of plastic esophageal tubes.

PLAN OF TREATMENT

All patients with carcinoma of the esophagus are carefully screened under the premise that resection and primary intrathoracic esophagogastrostomy is the procedure of choice. Contraindications are peripheral metastases, invasion of the tracheobronchial tree, advanced uncorrectable associated disease, disabling age and debility, and locally non-resectable lesions.

Bronchoscopy is performed on every patient, as well as esophagoscopy, to determine if bronchial extension of tumor is present. Occasionally a decision against surgery will be reached before endoscopy is performed. If so, endoscopic esophageal intubation is attempted at primary esophagoscopy. More often, intubation is decided upon because of either total or almost total esophageal obstruction when the patient is first seen at a far advanced stage; or to maintain swallowing of oral secretions and liquid diet during the anticipated period of obstructing edema following radiation therapy.

We have limited the use of esophageal dilatation to those patients who have completed a course of radiation therapy and in whom further reduction in the esophageal lumen has occurred either due to postradiation fibrosis or to late recurrence of cancer.

TECHNIC OF POLYETHYLENE INTUBATION

Tubes are easily formed by flanging one end of polyethylene tubing* after heating slightly with a match. The tubes measure approximately 0.6 cm. in external diameter with an internal diameter of 0.5 cm. and are cut into varying lengths. The proximal (upper) end is marked by wrapping several turns of .005 stainless steel wire distal to the flange. The tubes are sterilized by soaking in aqueous zephiran® chloride solution.

General anesthesia is most commonly used with an endotracheal tube in place because the instrumentation may be prolonged and distressing to the patient if performed under topical anesthesia. The 9 or 10 mm. by 45 cm. esophagoscope is passed, except in the presence of very high lesions when a shorter instrument is more easily used. The lesion is inspected and retained secretions and food removed. The direction of the residual lumen, if any, is estimated from the preoperative lipiodol or barium swallow and a small, reasonably flexible, Jackson dilator is carefully insinuated through the lumen.

* Clay-Adams "Intramedic" polyethylene tubing No. PE 410.

The length of the strictured area is then estimated and a tube selected, cutting the end obliquely with scissors to the correct length. The end is rounded to minimize the possibility of perforation. With the flanged end uppermost, the tube is passed through the esophagoscope over a previously inserted No. 10 or 12 French Jackson dilator and gently pushed along with a suction tip. (Fig. 1.) With the inlying dilator acting as a guide, the tube is gently pushed into the strictured area by advancing the tip of the esophagoscope against the flange until it rests on the protruding tumor mass. Occasionally a foreign body forceps is needed to place the tube accurately. The dilator is withdrawn and sterile saline washed through the esophagoscope to test the patency of the tube. The esophagoscope is withdrawn and the patient is allowed nothing by mouth on the day of the operation. The following day a lipiodol swallow is obtained under fluoroscopic observation with spot films to note the tube position and to hunt for a possible perforation. If all is satisfactory, the patient is fed fortified liquids and purées with no solid food allowed.

In occasional patients the tube has slipped and passed through the intestinal tract requiring replacement. Rarely a patient has plugged the tube with solid food requiring esophagoscopy for removal.

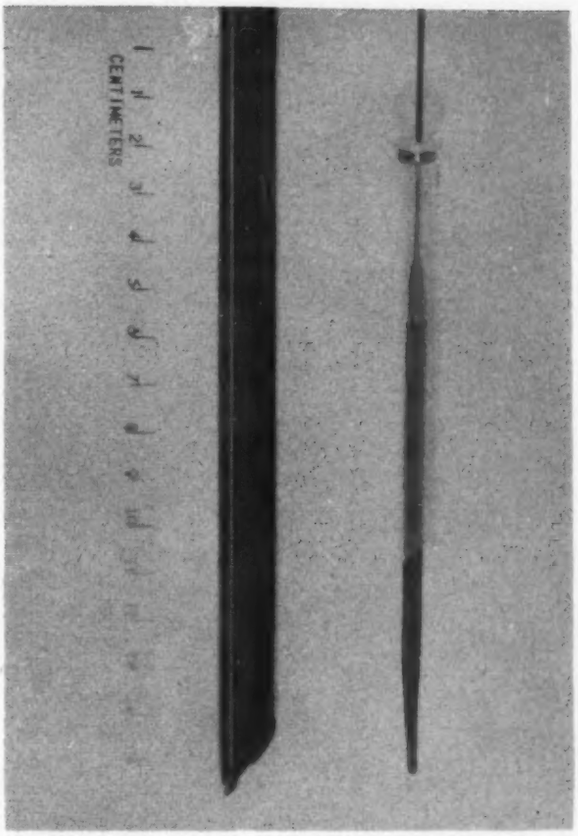

FIG. 1. Photograph of standard 9-40 Jackson esophagoscope next to polyethylene tube of usual size. The plastic tube is passed over a No. 12 French Jackson dilator as shown. Note loops of wire beneath proximal flange of tube for radiographic localization after insertion.

RESULTS

During the period 1950 to 1956, polyethylene intubation of the esophagus was considered in twenty-four patients and carried out in nineteen. The lesions were situated from the proximal esophagus in the thoracic inlet to the distal esophagus obstructed by ascending adenocarcinoma of the gastric cardia. Thirteen of the intubated patients thereafter swallowed liquids and purées satisfactorily and survived from one to thirty-six months. (Fig. 2.) Several of these patients remained well following completion of radiation therapy and removal of the tubes. The longest survivor without radiation was a sixty-eight year old man with an obstructing epidermoid carcinoma at the thoracic inlet who lived for one year, swallowing well, before dying of heart disease. The longest survivor following radiation therapy is a fifty-nine year old man with an obstructing lesion, probably lymphosarcoma, in the upper third of the esophagus. Intensive radiation treatment was given, during which time his nutrition was maintained by swallowing liquids through the tube. The tube was later withdrawn and the patient is apparently completely well and eating a normal diet thirty-six months later. (Fig. 3.)

The rapid improvement and weight gain in a semi-moribund cachectic patient who is again allowed to swallow is most dramatic. One of the most gratifying aspects is the elimination of overflow aspiration and the rapid healing of pneumonia which is frequently present.

COMPLICATIONS

In five of the twenty-four patients no lumen could be identified in the massively obstructing far advanced cancer; intubation was therefore not attempted. In six of the remaining nineteen patients tubes were inserted but did not function satisfactorily. One of these was simply angulated due to obstructing tumor and was

FIG. 2A. Roentgenographic spot film illustrating an inoperable adenocarcinoma of the stomach ascending the esophagus in a seventy-two year old man (C. F.). Barium swallow on admission to hospital.

FIG. 2B. Same case as Figure 2A. Lipiodol swallow following endoscopic insertion of plastic tube. Note wire loops on proximal end of tube just above diaphragm and reduction in size of dilated esophagus. The patient survived for two months, swallowing liquids satisfactorily.

removed, with no change in the patient's condition.

As might be expected in patients with massively invading "frozen" carcinomas of the esophagus with no lumen visible either on barium swallow or at direct esophagoscopy, introduction of a dilator may cause a perforation. We have taken the position that this risk is justified in patients with total esophageal obstruction and a prognosis measured in days or weeks. Actual esophageal perforation with the dilators or tubes has occurred in five patients. One recovered uneventfully only to die of the carcinoma shortly afterward. In four of the patients with esophageal perforation mediastinitis and/or pneumothorax developed and they died within five days to one month. In only one case was thoracostomy drainage of the pleural space required.

DILATATION

Following a satisfactory course of radiation therapy and after the edema has subsided, the tube is usually removed and dilatations are instituted if clinically significant stenosis is present. Often it is possible to use Hurst mercury bougies under topical pontocaine® anesthesia up to about the size of No. 38 French. This will allow a satisfactory soft solid diet to be taken. In occasional patients postradiation fibrosis develops so severely that Jackson-Plummer dilators are required. These are passed over a previously swallowed thread under general anesthesia. In successful cases mercury bougie dilatation may be needed as infrequently as once every two months.

Dilatation has been used in only five patients but has been effective in every case.

SUMMARY

The technic of endoscopic insertion of polyethylene tubes for palliation of obstructing esophageal carcinoma has been described.

FIG. 3A. Roentgenographic spot film of upper third of esophagus in a fifty-nine year old patient with proved malignancy (B. B.). Barium swallow before esophagoscopy.

FIG. 3B. Same case as Figure 3A. Lipiodol swallow after endoscopic insertion of polyethylene tube. The patient swallowed liquids well throughout total period of radiation therapy and the tube was withdrawn. The patient is clinically well with no roentgenographic evidence of recurrence three years later.

Tubes were inserted in nineteen of twenty-four patients in whom it was considered. Successful palliation was achieved in thirteen and failure was encountered in six patients. In five of the latter group complications developed which, together with the advanced state of the tumors, resulted in death between five days and one month later. The patients who swallowed liquids and semi-solids satisfactorily lived from one to twelve months, and one patient is well thirty-six months after intubation and radiation therapy.

The abrupt change from terminal illness with aspirational pneumonia to normal tem-

perature, adequate oral hydration and swallowing comfort is most dramatic in the successfully intubated patient.

Esophageal dilatation has also been used to maintain a lumen adequate for solid foods in patients who have completed a course of radiation therapy and in whom narrowing due to fibrosis or a recurrent radiation-resistant tumor subsequently develops.

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Rupture of the Esophagus

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RUPTURE of the esophagus occurs more frequently today because of the increased use of therapeutic and diagnostic endoscopy. It carries a very high mortality and morbidity unless early diagnosis and prompt therapy are instituted. The focal point in rupture of the esophagus is the contamination of the virginal fascial planes of the cervical and thoracic mediastinum with anaerobic bacteria of the mouth and irritating fluids of the stomach. Therapy must be directed toward the potential or actual infection that will follow.

Anatomically, the progress of infection after rupture is contingent upon the relationship of the esophagus to the fascial planes of the cervical and thoracic mediastinum. Therefore, knowledge of the anatomy of this region is of practical importance. (Fig. 1.) The deep fascia of the neck has four important component layers. These are: (1) the fascia of the carotid sheath, (2) the buccopharyngeal fascia, (3) the pretracheal fascia and (4) the prevertebral fascia.

The prevertebral fascia extends from the base of the skull to the third or fourth thoracic vertebra and covers the prevertebral muscles, forming the posterior wall of the retrovisceral space. The pretracheal fascia extends from the hyoid bone into the thorax to blend with the upper portion of the posterior pericardium and covers the trachea and thyroid gland, forming the anterior wall of the pretracheal space. The buccopharyngeal fascia extends from the base of the skull to the lower end of the pharynx and covers the constrictor muscles and esophagus, forming the anterior wall of the retrovisceral space. Laterally, it forms the posterior wall of the pretracheal space. The carotid sheath extends from the base of the skull to the root of the neck and covers the carotid arteries, jugular veins and vagus nerves. It is formed by a lateral extension of the pretracheal fascia, blending posteriorly with the prevertebral fascia [17].

These fascial layers divide the neck into three potential spaces that may communicate with the upper mediastinum. The pretracheal space lies between the superficial and pretracheal layers, the visceral space between the pretracheal and buccopharyngeal layers, and the retrovisceral space between the buccopharyngeal and prevertebral layers.

The retrovisceral space can be demonstrated to extend from the base of the skull to the sixth dorsal vertebra and is the most important pathway for the spread of infection between the upper cervical region and the mediastinum. The visceral space, containing the thyroid gland, trachea and esophagus, communicates directly with the posterior mediastinum. The pretracheal space is related inferiorly to the anterior mediastinum but does not communicate directly, for the pretracheal fascia merges below with the posterior pericardium and sends a dense fibrous layer forward and downward to be attached to the posterior surface of the sternum, thereby limiting the anterior compartment of the neck from the anterior mediastinum. The pretracheal space is also an important route, as infections from perforations through the pyriform sinus [42] and anterior wall of the esophagus may spread downward to reach it.

Anatomically, the location of the esophagus

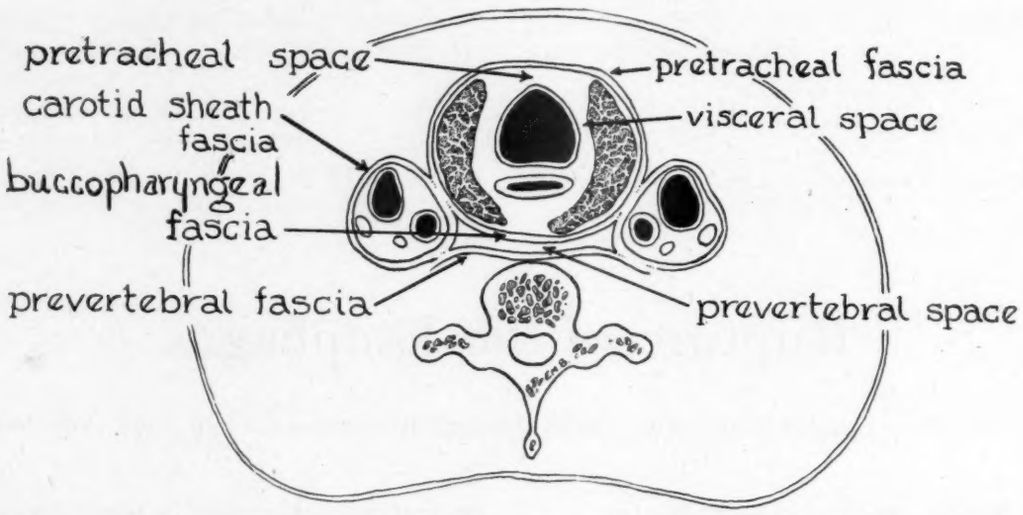

FIG. 1. Transverse section of the neck at the level of the sixth cervical vertebra. This demonstrates the four fascial layers delimiting the principal communicating pathways between the cervical and thoracic mediastinum.

makes it vulnerable to injury in many intrathoracic operations comparable to the injury of the common bile duct in upper abdominal procedures. In addition, the intrinsic anatomy of the thorax is a determining factor of the site of internal injury to the esophagus, for there are three normally narrowed areas of the esophagus as it traverses the neck and mediastinum. The first is the pharyngoesophageal orifice at the level of the cricoid cartilage; the second is the level of the aortic arch as it crosses the left main stem bronchus; and the third is the level of the lower esophagus at the diaphragm.

Morphologically, the lower esophagus is the weakest portion, for it tapers in a conical fashion and the muscles, being thinner than those of the stomach, are relatively weaker. Segmental defects with potential areas of weakness at the site of entrance of vessels and nerves through the muscle wall have been described [32]. The esophageal wall at this level is in direct contact with the mediastinal portion of the pleura, for the esophagus deviates slightly to the left, angulating anteriorly over the crus of the left diaphragm. Therefore, in rupture of the lower part of the esophagus direct entry into the left pleural space is more common.

The mechanisms are fourfold by which the esophagus may be ruptured. First, the entire wall may be immediately penetrated through all its layers either from within as by a sharp-pointed foreign body or from without by a missile injury. Second, the defensive mucous membrane may be lacerated, resulting in localized suppuration which leads to secondary periesophagitis and abscess formation. Third, the wall may be gradually eroded from within, as by a blunt foreign body or from without as by an aneurysm. Fourth, if a localized segment of the esophagus is deprived of its blood supply secondary to intrathoracic operations, the muscle layers will necrose and with subsequent suppuration a delayed rupture will occur [33].

The physiopathology of the events that follow rupture of the esophagus is basically the same, regardless of mechanism or causative factors. With penetration of all layers of the wall, anaerobic bacteria and irritating fluids are suddenly introduced into a new environment. Local defenses are incapable of meeting the rapid destruction of tissue which results in abscess formation with a wide surrounding zone of cellular inflammation. It is not the rupture *per se* that threatens the patient, but the consequence of introducing anaerobic infection into virginal fascial planes. The infection spreads rapidly and extensively and is favored by the fact that the retrovisceral space and superior mediastinum have practically no tissue barriers except for some loose areolar tissue and communicate with each other. The contents of the retrovisceral space and esophagus are aspirated into the mediastinum during the negative pressure phase of respiration and swallowed air is forced through the tear of the esophagus, inflating the spaces so that they are opened to easy passage of bacteria-laden saliva. In addition, the motion of the heart and

great vessels and the peristaltic action of the esophagus discourages the formation of the limiting plastic membrane [7].

Etiologically, there are a large and variable number of causes regardless of the basic underlying physiopathology. These causative factors are as follows: (1) external violence, (2) external infections, (3) external neoplasms, (4) aortic aneurysms, (5) penetrating missiles, (6) foreign bodies, (7) instrumentation, (8) increased intra-abdominal pressure, (9) tumors of the esophagus and (10) esophagitis.

External violence, as a cause of rupture, is rare and is almost always associated with serious injury to other viscera and is found incidentally during thoracotomy for other reasons [28,43,45].

An aortic aneurysm or external neoplasm, such as bronchogenic carcinoma of the lung, may erode the esophagus [3,20,24,45]. External infection may follow from trauma during difficult operative procedures in which the blood supply is compromised and a delayed perforation results [33].

Penetrating missile wounds may be caused by bullets, knives, ice picks and the like. In civilian practice these are more commonly seen to involve the cervical esophagus; militarily, thoracic esophageal injuries by such agents generally have other associated lesions.

Foreign bodies such as bones, dentures, pins or gastric tubes and the like may cause either sharp puncture wounds with immediate rupture or pressure necrosis with slower penetration. Anatomically, these commonly lodge at the level of the aortic arch, crossing the left main stem bronchus. Foreign bodies are one of the more common causes of perforation. Mathewson et al. [40] in reviewing fifty-four cases of traumatic rupture, found this to be the etiologic agent in fifteen cases (28 per cent).

Instrumentation through endoscopy or bouginage remains the most frequent source of injury [5,7,11,23,36,40,44]. Jemerin [21] reported an increase of more than 100 per cent in the decade ending in 1946, as compared with the decade before 1936. Seybold et al. [42] found that in the first thirty-six years of the Mayo Clinic there were twenty-one cases, while during the next six years, ending in 1949, there were twenty-nine cases. Mathewson [40], in studying fifty-four cases of traumatic rupture in San Francisco Bay hospitals during the past ten years, reports that one-third were due to esophagoscopy and more than one-half were due to instrumentation of some sort. Interestingly, in recent years, gastroscopy has been reported as the instrument causing the next largest number of instrumental injuries [7,42,44]. With a diseased esophagus perforation by instrumentation usually occurs at the level of an obstructing tumor or stricture. The most common location for rupture during esophagoscopy is at or just below the pharyngoesophageal orifice, which is the narrowest site of the three normally narrow areas of the esophagus [7,35,40,44]. Rupture at this level is usually posterior and the mechanism for this has been blamed on a transverse fold of mucous membrane across the posterior wall, simulating a physiologic sphincter, which may obscure the lumen and confuse the endoscopist [42]. Also, with the head in the position of hyperextension, the normal anterior convexity of the cervical spine is exaggerated and the rigid esophagoscope crushes the wall of the esophagus on the anterior wall of the bodies of the sixth and seventh cervical vertebrae. In many instances, these are osteoarthritic and have hypertrophic spurs on the anterior margins of the bodies [3,42,44]. Occasionally perforations at the esophagopharyngeal level may extend laterally or anterolaterally into the pyriform sinus [21,42]. The second most frequent site of instrumental rupture, usually occurring during dilatation for stricture, is in the lower esophagus just above the diaphragm.

An increase in intra-abdominal pressure is the common denominator in the many reported cases [23,24,37] of "spontaneous rupture" associated with other entities and such as might occur in pregnancy [47], labor [22], the act of defecation [51], strenuous exercise [18], epileptic seizure [25] or the severe vomiting with intracranial disease or peritoneal irritation [13,29]. A great deal has been written about this so-called "spontaneous perforation" or postemetic rupture in the past few years. It is most common in males between the ages of thirty-five and fifty-five. Many of the patients give histories suggestive of previously existing upper gastrointestinal tract disease, peptic ulcer, alcoholism or poor nutrition [5,24,29,31]. Wangensteen [48] believes that the word "spontaneous" should not be used. He prefers the phrase "acid peptic perforation" of the esophagus. Anderson [5] states that almost all cases show some evidence of pre-existing disease of

the esophagus and, in addition, there is some disturbance of the protein metabolism which may weaken the wall. He reports four cases with low total proteins and a reversed A/G ratio. Mallory and Weiss [29] first pointed out that the pressure changes occurring during the act of vomiting cause fissures in the mucous membrane at the cardiac end of the esophagus, and the local corrosive effects of alcohol and gastric juices result in linear ulcerations of the mucous membrane with subsequent infection and abscess formation, setting the stage for rupture through the remaining wall. Fleischner [14] believes that these Mallory-Weiss lacerations result from the disassociation of the external and internal esophageal hiatal sphincters and represent incomplete ruptures. Thus, with vomiting or the application of an external force to the abdomen, a sudden rise occurs in the intra-abdominal pressure. When this pressure is transmitted into the esophagus, rupture will occur. The spontaneous and postemetic ruptures reported in the literature occurred at the lower end of the esophagus following violent retching or vomiting (i.e., increased abdominal pressure) and many reports [5,9,28,38,40] point out that it is an organ rupture and should therefore be considered traumatic in origin.

Clinically, all of the aforementioned etiologic factors are important, as well as the history of the speed of onset, duration and location of the predominant symptoms and signs. These facts determine the approximate size, location and severity of insult and assist in ascertaining the correct plan of management for the particular situation.

In the history previous endoscopy, the swallowing of a foreign body, missile injury or vomiting are of particular importance. In addition, the many other etiologic agents previously mentioned should be searched for, as well as the time since onset. In the "spontaneous ruptures" the onset is sudden and a history that is suggestive of previous upper gastrointestinal peptic disease along with alcoholic intake and vomiting is important [5,14,19,29].

The early symptoms may be almost negligible and this is particularly so with small ruptures or those that occur slowly. If the rupture is cervical, pain located in reference to the neck, "throat" or upper anterior thorax is the most prominent feature. It can be quite severe and is aggravated by motion to such an extent that the patient holds his neck rigid. There may be dysphagia and dysphonia. If the rupture occurs in the lower esophageal region, as in the postemetic type, pain is the first and most common symptom to appear. It is usually quite severe, may be aggravated by motion and is substernal or epigastric in location. It may radiate straight through to the back. If the pleura is penetrated, there will be pleurisy on the affected side. Often the patient will give a bizarre history of something bursting and moving around in his chest [1,31]. In the case in which the pleural space is opened, there will be respiratory symptoms; with massive contamination of the mediastinum, there will be other symptoms related to the peripheral vascular collapse.

The signs will vary depending upon the location and mechanism of perforation and the stage at which the patient is examined. In lower esophageal rupture with rapid mediastinal and pleural soiling, the patient may quickly appear severely ill, dyspneic, tachypneic and cyanotic. Although at the onset the temperature may be elevated only a degree or so, it rapidly rises. Subcutaneous emphysema is the earliest sign, appearing first in the suprasternal notch and spreading into the neck and down over the thorax. With pleural penetration, signs of pleural effusion or hydropneumothorax are present. Violation of the pleura surgically as well as by inflammation or irrigating gastric fluids is a common cause of upper abdominal muscle spasm and ileus [4], and these may be present. Occasionally the severe muscular spasm has been reported to lead to exploratory celiotomy for a ruptured abdominal viscus [24,42]. If the rupture has occurred in the neck there may be no early signs, particularly if it is small. With a larger tear or at a later stage there will be some local tenderness, induration and crepitus from the subcutaneous emphysema.

The oral hygiene during the course of the physical examination should be noted, for if it is good, as is usually the case in edentulous persons, it may have a bearing in the later management and prognosis.

Laboratory examination, in the early phase, may show a leukocytosis with an increase in the polymorphonuclear cells, but the changes in the hematocrit and hemoglobin determinations develop later and reflect the dehydration.

The x-ray findings will depend upon the site and nature of the perforation. Early in the course there will be subcutaneous emphysema. In a composite study [37] of thirty-five cases with thoracic esophageal rupture, 75 per cent of the patients had hydro(pneumo)thorax with or without subcutaneous emphysema, and only three cases had a negative posteroanterior chest x-ray, of which one showed retropharyngeal air on the lateral x-ray only. In no case was there subdiaphragmatic air. Other x-ray findings may be a widening of the superior mediastinal shadow with an air and fluid level. The trachea may be displaced anteriorly from an accumulation of inflammatory products in the retrovisceral space, as shown on lateral x-ray. This normally narrow space is increased to more than the width of the trachea [3,11,20]. Radiopaque substances, such as lipiodol or dionosil (barium should *not* be used) with fluoroscopic control or x-ray may demonstrate the site of the rupture.

Thoracentesis with the recovery of air or fluid that may be examined for food particles, hydrochloric acid or blood is helpful. The patient may be given an oral dye that can be recovered in the pleural fluid which is diagnostic of an esophageal fistula.

The diagnosis is most promptly made by thinking of esophageal rupture, for a high index of suspicion is the most important single factor in the early diagnosis. Any patient who has had instrumentation, probable ingestion of a foreign body, or who has been vomiting and develops pain referable to the neck or thorax with subcutaneous emphysema should be considered to have an esophageal rupture until proved otherwise.

Various diagnostic triads have been emphasized [2,6,28], of which the salient features are vomiting, substernal or epigastric pain, respiratory symptoms, abdominal spasm, subcutaneous emphysema and leukocytosis. These symptoms and signs may be masked by the usual postoperative sequelae and general use of antibiotics whenever unrecognized injury [33] to the esophagus occurs during a major intrathoracic procedure. The diagnosis in these cases may be most readily made by having the patient swallow a dye and recovering it in thoracentesis fluid, or observing the course of swallowed radiopaque substance under roentgenographic control.

The differential diagnosis of esophageal rupt re includes the consideration of a number of entities depending upon the site of rupture. In the neck, esophageal rupture may be confused with acute tonsillitis, thyroiditis, peritonsillar abscess or laryngeal edema. When the signs and symptoms are predominately thoracic, spontaneous pneumothorax, coronary thrombosis, dissecting or ruptured aneurysm or a pulmonary embolization must be considered. With abdominal signs, viscus rupture, intestinal obstruction, pancreatitis, splenic infarction or mesenteric thrombosis must be thought of. Barrett [6] states that the diagnosis may depend upon who sees the patient first, for the internist is likely to diagnose the case as myocardial-infarction, while the surgeon may diagnose it as a perforated peptic ulcer.

In general, if subcutaneous or mediastinal emphysema is present, one may safely rule out the various inflammatory processes related to the neck, coronary thrombosis, aneurysm rupture, pulmonary embolization, intestinal obstruction, volvulus, mesenteric thrombosis and splenic infarct. Acute pancreatitis or perforated abdominal viscus may occasionally cause a pleural effusion, but do not cause subcutaneous tissue air. These entities would have a history of upper gastrointestinal disturbance, and an elevated serum amylase would distinguish pancreatitis. A perforated abdominal viscus would cause subdiaphragmatic air, demonstrated by x-ray. This does not occur in rupture of the esophagus. A pneumothorax, which is not secondary to esophageal rupture, is not accompanied by the severe systemic collapse that is caused by the sudden massive outpouring of the irritating fluids and bacteria into the pleural cavity. Rupture of a previously herniated stomach or bowel into the mediastinum or pleural cavity may simulate rupture of the esophagus and this is most easily differentiated by having the patient swallow a benign radiopaque substance under x-ray or fluoroscopic observation.

The prognosis is related to the site, etiologic agent, size and elapsed time of the rupture. If the perforation is cervical and of small size and recognized early, the prognosis is good. If the rupture is large and in the thoracic mediastinum and recognized late, the prognosis is grave. In the acute stage nearly all deaths are due to shock secondary to the hypoxia from tension pneumothorax and overwhelming sepsis. Later deaths are due to the

systemic effects of chronic sepsis and fistula formation with malnutrition. Jemerin [*21*], reviewing the result of sixty-nine treated cases, states that the mortality prior to 1939 was 77.3 per cent, whereas the mortality between 1936 and 1948 was 17 per cent. He believes that the most important factor instrumental in reducing the mortality is the use of prompt surgical intervention. Of fifty-three patients who died in an era prior to the general use of antibiotics, Kinsella [*24*] found that thirteen died in less than twelve hours, twenty-four died in less than twenty-four hours and eight died in less than forty-eight hours. Almost all of these reported cases occurred prior to the era of intensive antibiotic therapy, as a part of the management in each case, and the prognosis in all patients with varying types of ruptures has greatly improved since the institution of specific antibacterial therapy.

The therapy of esophageal rupture is manifold, as is evidenced by the varied opinions of many authors in the recent literature [*7,21,23, 24,30,31,35,40,49,50*]. Some authors advocate an immediate direct surgical intervention [*5,6,8,9,27,28,38,42,46*], while others prefer a more conservative approach [*2,11,12,23,34,41*].

Inasmuch as many of these reports deal with different modalities of the same subject, it is apparent that there is no one single procedure of choice in the treatment of esophageal rupture. In choosing the definitive therapy, consideration must be given to the approximate size of the rupture, its location, the time that has elapsed since the onset of pathologic changes, the etiologic agent responsible, the patient's systemic reaction to the insult and the state of his oral hygiene.

In the past, rupture of the alimentary canal in any of its portions has predicated the use of certain general over-all principles of débridement, drainage and diversion as needed. However, in recent years the management of hollow viscus rupture anywhere in the body has been greatly augmented by the use of appropriate antibacterial therapy.

It is in the small cervical esophageal ruptures in the early stages that this form of therapy has its greatest value. These minor tears (mucous membrane or entire wall) may ensue following endoscopic examination or erosion by a foreign body. These patients, when seen immediately or when without evidence of gross contamination, may be most effectively treated with large doses of broad-spectrum antibiotics administered early and a regimen of nothing by mouth supplemented by intravenous alimentation. Mathewson et al. [*40*], in receiving fifty-four cases of esophageal rupture of which twenty-one were cervical, point out that when conservative management was the treatment of choice there were no deaths in eleven cases of conservatively treated cervical rupture. However, in the conservative management of mid- and low thoracic ruptures, five of fourteen such patients died. Thus, ruptures above the pleural reflection are more amenable to conservative therapy than those below.

One important factor in the conservative management of rupture of the esophagus that has largely been overlooked in the past is the status of the patient's oral hygiene. In the crevices of the gingival margin both aerobic and anaerobic bacteria are harbored in spite of attempts to cleanse the teeth. Even with a restricted oral intake, the patient continues to swallow his own saliva and bathes the site of rupture with the anaerobic bacteria so catastrophic to the virginal fascial planes of the mediastinum. Therefore, it is highly recommended that the patient be given broad-spectrum antibiotic lozenges to dissolve in his mouth. Edentulous persons have such a harmless bacterial flora in their mouth that in borderline cases conservative management may be the procedure of choice on this basis alone.

Large cervical esophageal ruptures in the early stages may be most successfully treated by primary suture. If the esophagus is severed by a missile injury primary end-to-end anastamosis may be considered [*15*]. The neck may be explored by extension of the original wound, but a collar incision gives excellent exposure and is recommended for the most thorough exploration of the neck. Overstreet and Oschner [*35*] recommend a standard oblique sternomastoid retracting incision under local anesthesia with primary closure of the rent. Nonabsorbable material and a transverse inverting technic are used. Drains should be placed away from the line of repair and the patient kept in the head-down position for the first forty-eight to twelve hours to facilitate drainage from the upper mediastinum. Clear liquids are begun by mouth after twenty-four hours.

Large or small cervical esophageal ruptures in the late stages when there has been cervical

mediastinal contamination with abscess formation may be most effectively treated by classic cervical mediastinotomy. This will drain the retrovisceral space of the neck and the superior posterior mediastinum. If an abscess is not discovered in the retrovisceral space, it may be possibly found in the pretracheal space [*42*].

Large and small ruptures of the thoracic esophagus (i.e., involving the pleura) in the early stages are best treated with early open operation [*5,6,27,28,38,40*]. If the rupture involves the mediastinum alone, recovery has been reported to occur without surgical intervention [*39,40*]. At the time of open thoracotomy, the mediastinum and pleural cavity are thoroughly cleansed mechanically and with saline irrigation, the rent is repaired in a manner similar to a standard esophagotomy, and the Levin tube is passed for stomach decompression. The area is bathed with local antibiotics and the lung is actively re-expanded. The patient is given nothing by mouth for a few days, except for oral antibiotic lozenges; systemic antibiotics and intravenous alimentation are administered.

Large or small esophageal ruptures of the thoracic esophagus in the late stages, in which mediastinitis with abscess formation and empyema have developed, may be drained by the use of a standard posteroinferior mediastinotomy, a closed drainage of the pleural space for the empyema or a combination of these.

In delayed traumatic rupture of the thoracic esophagus a staged procedure may be necessary. These cases are more difficult because of the indurated nature of the tissue in this area, and a multiple layer closure is difficult. A thickened flap of pleura can be used to reinforce the closure, as the apposition of an expanded lung greatly contributes to the permanency of the closure. A modified thoracoplasty may be needed if the lung is not present.

Other special cases may arise in which the etiologic agent or time factor may dictate the definitive form of therapy to be employed. The use of closed thoracotomy, bilateral empyema drainage, thoracentesis and the intrapleural administration of antibiotics have all been reported in particular circumstances [*1,12,34,41*]. Overstreet and Oschner [*35*] report a case, with recovery, in which a rupture occurred through the site of a stricture at the esophageal hiatus, necessitating resection and primary esophagogastrostomy.

CASE REPORTS

The four typical cases to be presented demonstrate the usual history, clinical findings and course in rupture of the esophagus. The poor prognosis in the era prior to the general use of antibiotics and definitive surgery is contrasted with the more optimistic outcome of present day therapy. The possibility of conservative management in selected cases is shown.

CASE I. G. E. (Roosevelt Hospital No. 17791), a sixty-one year old man had eaten a very large Thanksgiving Day meal. Sudden epigastric pain developed with vomiting, and he later became dyspneic and orthopneic. There was no previous contributory history. On physical examination, the temperature was 100°F., pulse 144, respirations 20, and blood pressure 150/100. The patient was cyanotic, dyspneic, cold and clammy. There was evidence of a left hydropneumothorax with a good deal of generalized abdominal rigidity with epigastric tenderness. The white blood count was 11,900 with 60 neutrophils. X-ray showed a massive atelectasis of the left lung with pneumothorax, a fluid level to the sixth intercostal space and shift of the mediastinum to the right. It also demonstrated subcutaneous emphysema in the neck. The patient underwent emergency thoracentesis with the removal of 350 cc. of brown odorous fluid. However, before further therapy could be undertaken, he rapidly went into profound shock and died. Postmortem examination revealed a rupture of the esophagus approximately 3 cm. in length, on the left posterolateral side, 1 cm. above the stomach, with no surrounding inflammation. There was a left hydropneumothorax with massive atelectasis of the left lung and emphysema of the neck and chest.

Comment: This case, which occurred in the era before the general use of antibiotic therapy, demonstrates the catastrophic nature of rupture of the esophagus and shows the classic and clinical features of this disease.

CASE II. F. K. (Ft. Hamilton Veterans Hospital No. 28783), a sixty-one year old man with a previous history of upper abdominal pain and massive upper gastrointestinal bleeding, was undergoing a complete general work-up. Esophagoscopy was performed with a 9-45 instrument and with the patient under

FIG. 2. Case II. Subcutaneous emphysema of the neck and right total pneumothorax secondary to traumatic rupture of the esophagus by esophagoscopy.

topical cocaine anesthesia, at which time recognized difficulty and bleeding were encountered at the level of the cricopharyngeus. No other disorder was found. At the time of esophagoscopy the patient complained of chest pain and esophageal rupture was considered, as became evident when emphysema of the neck and a right pneumothorax developed. (Fig. 2.) Lipiodol was given by mouth and demonstrated a fistula into the right pleural cavity. A thoracentesis was performed to remove the air. Approximately twelve hours after injury cervical mediastinotomy was performed, at which time a 1 cm. long longitudinal slit was found 3 cm. below the cricopharyngeus muscle, together with suppuration in the retrovisceral space. A toilet was done and local antibiotics were dusted in the area; drains were placed into the superior mediastinum. A Levin tube was passed into the stomach and an intercostal tube was placed in the right chest. Some atelectasis of the right middle lobe developed, and on the third day after the original esophagoscopy the patient vomited a large amount of coffee-ground material. Massive upper gastrointestinal bleeding continued as manifested by coffee-ground material and bright red blood draining through the Levin tube. He became progressively lethargic and died ten days after the original esophagoscopy. Postmortem examination revealed a 1.5 cm. cervical esophageal rupture which was apparently healing. There were varices of the lower esophagus and several peptic ulcerations. The stomach and duodenum were distended with blood and there was an acute perforation of the first part of the duodenum with several superficial duodenal ulcers. The pathologic diagnosis was duodenal perforation with localized peritonitis and exsanguination. No mediastinitis was noted.

Comment: If injury to the cervical esophagus is suspected, due to instrumentation, there may be no signs or symptoms early in the course of the disease. In this patient, however, pain developed early along with emphysema of the soft tissues of the neck. X-ray examination of the neck with lateral and oblique views can promptly detect this even if not detected by physical examination. With such evidence of a large rupture in the cervical esophagus, at an early stage, with evidence of pleural violation and severe systemic reaction, open operation should be performed immediately.

CASE III. J. F. (Doctors Hospital No. 79875), a sixty-nine year old woman undergoing a work-up for progressive dysphagia, underwent esophagoscopy with biopsy of a tumor at another hospital. The biopsy specimen was reported one day later as normal lung, at which time this obese patient complained of some shortness of breath, but otherwise felt quite well. She was admitted to the hospital as an emergency and physical examination, as confirmed by x-ray, showed a one-third right pneumothorax and no evidence of subcutaneous emphysema. The temperature, white blood count and all vital signs were normal and remained so for the next twenty-four hours. Following admission thoracentesis was performed, aspirating fluid and air and re-expanding the lung. The patient was placed on a regimen of nothing by mouth, intravenous alimentation, blood transfusions, penicillin and streptomycin, and oral penicillin lozenges. She continued to have a stable course on such management. One week later she was able to undergo esophagectomy for the neoplasm which was in the mid-esophagus at the level of the aortic arch. At that time a small opening on the right lateral wall in the mid-esophagus was found, with an area of trauma to the right lower lobe. The patient had a benign recovery from this major procedure.

FIG. 3A. Case IV. Rupture of lower thoracic esophagus secondary to gastroscopy; subcutaneous emphysema of neck and pneumomediastinum.

FIG. 3B. Case IV. Lipiodol swallow demonstrates an esophageal mediastinal fistula on the right side.

FIG. 3C. Case IV. On the second postoperative day early chemically induced pneumonitis was revealed. Note Levin tube for constant gastric decompression and chest catheter for active re-expansion of lung.

FIG. 3D. Case IV. Lateral roentgenogram taken postoperatively demonstrating the retention of lipiodol outlining the mediastinum.

Comment: Elderly edentulous persons with recognized esophageal rupture and no evidence of severe systemic reaction may be safely managed conservatively, particularly because of the availability of antibiotics.

CASE IV. V. O. (Ft. Hamilton Veterans Hospital No. 57114), a twenty-seven year old man was admitted with a history of epigastric pain and vomiting. He had undergone previous operation for perforated duodenal ulcer and continued peptic symptoms, and had had two episodes of massive gastrointestinal bleeding. As part of the diagnostic work-up, gastroscopy was performed without difficulty. One hour later right epigastric and lower chest pain with emphysema of the face and neck developed. His temperature quickly rose to 103°F., the pulse was 120 and the respirations were 28. Lipiodol swallow x-rays revealed dye outside of the esophagus on the right side about 8 cm. above the diaphragm. (Fig. 3.) There was air in the mediastinum outlining the heart and in the fascial planes of the neck. The white blood count was 12,700. Immediate thoracotomy was performed, at which time a 1 cm. perforation in the posterior wall of the esophagus was noted 5 inches above the cardia. There was mediastinal fat necrosis. A toilet was done and the rupture closed primarily with silk. The mediastinal pleura was left open and the lung re-expanded with underwater seal chest tube drainage. A Levin tube was passed. Postoperatively the patient had a stormy course of atelectasis and secondary pneumonitis, requiring repeated bronchoscopies. The Levin tube was removed on the third postoperative day and the patient resumed an oral intake. At the time of discharge the patient was considered to have fully recovered from the trauma, although the admitting diagnosis was still in doubt.

Comment: Patients with large ruptures of the lower thoracic esophagus in the early stages should undergo immediate exploration with débridement and repair of the rupture.

CONCLUSIONS

1. Historically, rupture of the esophagus has been known for over 200 years and the increasing number of reports in the literature in recent years statistically bears out the impression that rupture of the esophagus occurs more frequently today.

2. Pathologically, the contamination of virginal cervical and thoracic mediastinal fascial planes with anaerobic bacteria of the mouth and irritating fluids of the stomach results in the rapid destruction of tissue and a widely spreading infection consequent to the anatomy of the involved area. This is the focal point toward which therapy must be directed.

3. Experimentally and mechanically, so-called "spontaneous" rupture of the esophagus does not occur unless by some extraesophageal force that suddenly increases the intraluminal pressure. As it is an organ rupture, it should be considered traumatic in origin.

4. Clinically, there are many etiologic agents to be considered in eliciting the history, and the signs and symptoms will vary depending upon the size, site and time after rupture. The most common location of rupture of the esophagus is in the cervical esophagus and is most frequently secondary to instrumentation.

5. Diagnostically, the most important point is to think of esophageal rupture. The most important ancillary aid to diagnosis is the use of a benign radiopaque material or a dye to demonstrate an esophageal fistula.

6. Prognostically, the outcome is extremely guarded, but it has improved in recent years with prompt surgical intervention and antibiotic coverage.

7. Therapeutically, treatment must be based on the size and location of the rupture, the stage at which the patient is seen, the etiologic agent, the patient's systemic reaction to the insult and the status of his oral hygiene. Antibiotics are quite important in the over-all management of any patient and minor cervical recognized instrumental ruptures are amenable to conservative management, whereas large dramatic ruptures, especially with pleural violation, should have immediate direct repair and drainage. Shock which is due to the disturbed physiology of the body is not a contraindication to surgery, for the surgery itself is a resuscitative measure. Rapid and complete reexpansion of the lung is of paramount importance.

8. Oral hygiene should be given careful scrutiny, especially when conservative management is desirable. Edentulous persons may be conservatively treated with more safety than those whose gingival margins harbor the potentially dangerous anaerobic bacteria. Oral antibiotic lozenges and peroxide mouth washes are useful adjuvants in the therapy of these patients.

SUMMARY

The anatomy, pathology, etiology, clinical and laboratory manifestations, and therapy of rupture of the esophagus have been reviewed. Four cases have been presented. The relationship of the status of the patient's oral hygiene to the type of management to be employed is emphasized.

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Spontaneous Rupture of the Esophagus

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BY definition, spontaneous rupture of the esophagus must occur in a normal organ. It must be completely involuntary and without external force. Truly spontaneous rupture of the esophagus is not common. Some authors have suggested that this term not be used. Samson [*21*] suggested the term postemetic rupture. This accurately describes many of the cases included under spontaneous rupture. Wangensteen [*24*] prefers the term acid-peptic perforation of the esophagus; this is also accurate. Several of the cases included in this group are neither postemetic nor a result of peptic esophagitis. Fifty recently reported cases form the basis of this report.

This condition nearly always results from rupture, not perforation, of the esophagus. Most frequently it is due to increased intraluminal pressure within the esophagus. Occasionally there are changes within the esophageal wall resulting from peptic esophagitis or from other causes. The term spontaneous rupture is used here, not in its strictest sense but simply to include those cases of rupture of the esophagus that do not result from more obvious causes such as direct trauma, foreign bodies or instrumentation, or as a result of perforation due to neoplasm, inflammation or suppuration.

Far more important than the name of this condition is its early recognition and treatment. This article is written to stimulate interest in and an awareness of this condition.

History. Spontaneous rupture of the esophagus was first described by Boerhaave [*4*] in 1724. The first antemortem diagnosis was made by Schoenlein and reported by Meyer [*16*] in 1859. To Sencert [*22*] is accredited the first operative cure in 1911. Overholt [*8*] performed the first successful thoracotomy and pleural drainage for this condition in this country in 1943. Two additional cases were discussed by Graham [*11*] in 1944. Barrett [*3*] reported the first survival following thoracotomy and repair in 1946. By December, 1950, ninety-two cases had been reported, with eighteen survivals [*1*].

Many excellent reviews on the subject have been published. The more recent articles are those of Ware, Shnider and Davis [*25*] and of Mackler [*13*]. The most recent is an excellent article by Derbes and Mitchell [*8*]. Without an attempt to exhaust the literature thoroughly, nearly 200 cases of this condition have been collected.

This condition is being recognized by a greater number of physicians and interest is increasing. Derbes and Mitchell [*8*] point out that of fifty-five patients operated upon thirty-five survived. The mortality rate is improving but still remains much too high. This condition should therefore be kept in mind when considering acute emergencies. The value of early recognition cannot be overemphasized. Early diagnosis and adequate treatment are imperative to reduce the morbidity and mortality.

ETIOLOGY

From the etiologic standpoint these cases can be divided into the following three groups: those due to esophagitis and ulceration; those resulting from increased intraluminal pressure as a result of vomiting or other indirect force; and those that are apparently spontaneous, occurring in a normal organ and without obvious cause.

Of fifty cases, esophageal ulceration was found in eleven. Thirty-six patients had a history of vomiting. Four of these patients had pain followed with vomiting. There were nine cases in which the esophagus was described as normal and in which there was no history of vomiting or obvious cause for the rupture.

There is adequate experimental evidence to show that rupture of the normal esophagus can occur. That it does occur is evidenced by the fact that many such ruptures have been reported in organs which were grossly and micro-

scopically normal. Kinsella et al. [12] state that the average adult can expire with pressures up to 125 mm. Hg and strong men may attain a pressure of 190 mm. Hg or 3.63 pounds. Under certain conditions of stress the pressure may be several times that amount. No accurate figure is available.

McKenzie [15] in 1884 stated that pressures of 5¾ to 11 pounds, with an average of 7 pounds, are necessary to rupture the normal human esophagus. Burt [6] in 1931 determined that the esophageal muscle would rupture at an average of 3.49 pounds of pressure and the mucosa at an average of 4.07 pounds. Kinsella et al. [12] state that according to their experiments the normal human esophagus will rupture at pressures ranging from 10 to 33 cm. Hg, the average being 19.27 cm. Derbes and Mitchell [8] in a group of ten cases showed that the esophagus may rupture with less than 1 pound of pressure, the least being 0.45 pound and the greatest 46 pounds of pressure. In six of their ten cases the normal esophagus ruptured within the physiologic range, which is approximately 3.63 pounds of pressure.

The wide variation in the determined strength of the normal esophagus is due to several factors: (1) The esophagus of an adult will rupture under less pressure than that of a child [6]. The esophagus of a cadaver past fifty years of age has much less strength. (2) A sudden increase in pressure will cause rupture at a much lower pressure than a gradual increase, the rapidity of distention being the important factor [12]. (3) The time interval between death and the experiment is a factor. It has been pointed out that within an hour after death the strength of the esophagus may decrease as much as 50 per cent [8]. (4) The esophagus in its normal position will rupture with less pressure than the isolated esophagus. The esophagus being supported throughout most of its length is less able to dilate over these areas. This puts more pressure on the non-supported lower end, particularly on the left side.

Of nine cases in which the normal esophagus ruptured without vomiting or other obvious cause, eight patients were fifty-three years of age or older. One patient was a twenty-six year old man.

Wangensteen and his associates have done some most interesting experimental work regarding esophagitis and acid-peptic perforation of the esophagus. In a recent article [5] they have shown conclusively that esophageal perforation can be caused in dogs by ligation of the pylorus followed with intermuscular administration of histamine in beeswax. After pyloric ligation alone, esophagitis developed in all ten animals following an injection of histamine; perforation occurred in two. If a cervical esophagostomy is added, perforation occurred in ten of twenty-five. If the acid-secreting portion of the stomach is removed, no ulceration or perforation occurs. Spontaneous rupture does not occur in the dog since the bursting pressure of the esophagus is far above the physiologic range. These authors have shown that the esophageal mucosa is extremely sensitive to the gastric juice, the peptic activity of the gastric juice being the important factor [10]. In the experimental animal pancreatic juice and bile or a mixture of both causes digestion and destruction of living esophageal mucosa [7]. Removal of the acid-bearing portion of the stomach does not alter the effect of the pancreatic juice and bile upon the esophageal mucosa [2]. Secondary anemia is a result frequently of esophagitis [7]. Increase in the intracranial pressure has no effect upon the production of esophagitis. Islands of gastric mucosa rarely found in the esophageal mucosa may be the cause of esophageal ulceration and perforation [10].

This excellent work certainly proves that esophageal ulceration may often be caused by reflux of gastric juice, pancreatic juice or bile into the esophagus. It does not prove that all "spontaneous ruptures" of the esophagus are due to acid-peptic esophagitis. It is interesting to note that not a single case of rupture of the esophagus has been described in an adult with peptic ulceration and pyloric obstruction, even though severe vomiting may occur. Two cases have been reported in children, one with congenital pyloric stenosis and one with high intestinal obstruction. Another interesting fact is that esophageal perforation after pyloric ligation occurs in the upper third of the esophagus, not the lower third.

Increased intraluminal pressure within the esophagus plays an important part in rupture of the lower third of the esophagus. Vomiting is the most frequent cause of increased intraluminal pressure. Rupture may be caused by indirect trauma, undue straining or coughing, or any other movement or force that will cause

TABLE 1
PATHOLOGY (FIFTY CASES)

Esophagus		Position of Rupture				
Normal	Ulceration	Left Postero-lateral	Right Postero-lateral	Posterior	Anterior	Unknown
39	11	36	5	7	1	1

sudden increase in intraluminal pressure. Mosher [17] has pointed out significant anatomic features which cause rupture to occur in the lower third of the esophagus. These will be discussed later. The fact that these ruptures occur as a result of vomiting, coughing, defecation, etc., is significant. Of the fifty cases reviewed, thirty-six patients had vomited.

The part that malnutrition and starvation play in this condition is not known. It cannot be denied that many of these persons are chronically malnourished. It would be most interesting to determine the strength of the esophagus in chronically malnourished animals as compared to the normal animal.

PATHOLOGY

Rupture of this type is most frequently seen in the left posterolateral wall of the esophagus. It occurs most commonly in the lower 8 cm. of the organ. In a series of ten experimentally produced ruptures of the human esophagus reported by Derbes and Mitchell [8], eight occurred in the left posterolateral position, one occurred in both the left anterolateral and right anterolateral position, and one occurred in the right anterolateral position. In Mackler's series [13] sixty were on the left and two were on the right side of the esophagus. In McKenzie's report of experimentally produced ruptures [15] sixteen were on the left and two occurred on the right. Of fifty clinical cases, thirty-six ruptured in the left posterolateral position, seven were described as posterior, five as right posterolateral and one as anterior; in one case the position of rupture was not described. (Table 1.) The tear is usually vertical and may be from 2 mm. to 8 cm. in length. Occasionally it will involve the stomach wall and severe bleeding may occur. Perforation of the middle third of the esophagus has been described twice and complete disruption of the esophagus has been described on two occasions [19]. Transverse tears of the esophagus have been described twice [1].

Rupture of the esophagus has occurred in patients with poliomyelitis, meningitis, paresis, intracranial hemorrhage, pregnancy and brain tumor. Such ruptures have occurred during weight lifting, roentgen examination of the gastrointestinal tract, defecation and administration of anesthesia. Several cases have been due to indirect trauma such as blows to the abdomen [18]. Esophageal ulcers were found in eleven of fifty cases. (Table 1.) The others were described as normal. In only twenty-six of the fifty cases was a postmortem examination made.

Several explanations have been suggested regarding the persistent site of the rupture. It has been pointed out that the esophagus is supported on three sides by connective tissue extending between the vena cava and the azygos vein. Only on the left is the esophagus free to dilate and rupture. However, in the experimental perforations produced by Mackler [13] in which the esophagus was removed from its surroundings, the perforations occurred with regularity in the same position along the left posterolateral wall, suggesting that the cause for this is due not only to the relationship of the esophagus with other organs but also to intrinsic weakness within the esophageal wall. These anatomical weaknesses are best pointed out by Mosher [17], who made these points: (1) The esophageal muscle at the lower end terminates in a conical fashion, the tapered end being extremely thin. (2) Inherent weakness exists at the site of the entrance of vessels and nerves into the muscles at the lower end of the esophagus. (3) The muscles of the esophagus at the lower end are relatively weaker than the muscles of the stomach. (4) Segmental defects are found in the circular muscle layer at the lower end of the esophagus.

Derbes and Mitchell have suggested an

TABLE II
INCIDENCE (FIFTY CASES)

Sex	Race		Age (yr.)							
	White	Negro	1–10	10–20	20–30	30–40	40–50	50–60	60–70	70–80
Male	41	4	4	1	1	3	5	23	5	3
Female	2	3	1	0	0	2	0	1	0	1

explanation for the longitudinal character of these ruptures and have worked out a rather elaborate mathematical formula to prove their contention. By the use of the principles of free body diagram it is concluded that hydrodynamic forces compel longitudinal tearing of the esophagus. In experimentally produced rupture of the esophagus the muscularis gives way first, allowing the mucosa to balloon out through the rent in the muscular wall. It is the mucosa that remains intact, finally giving way with a loud report.

A similar condition has been described by Mallory and Weiss [*14*] and observed by these men in fifteen patients in whom massive gastric hemorrhage developed after an alcoholic debauch. Fissure-like longitudinal lesions of the mucosa 3 to 20 mm. long and 2 to 3 mm. deep were found in four of these patients. These lesions were arranged around the cardia. Mucosa-splitting lesions have been noted in pernicious vomiting of pregnancy. These heal, occasionally causing esophageal stricture.

CLINICAL HISTORY

Rupture of the esophagus is most commonly seen in men (85 to 90 per cent). A review of fifty cases showed five to be in women. The youngest reported patient was seventeen months of age and the oldest was seventy-five years old. The largest number of cases occur in patients between the ages of fifty and sixty. (Table II.)

Some of these patients appear to have enjoyed good health previous to the onset of this condition. Many, however, have a history suggestive of peptic ulcer; others are chronic alcoholics. Dietary indiscretion is common among these persons, and as a result they may be chronically malnourished. Esophageal rupture has occurred in persons in all walks of life and in some who have not practiced known dietary indiscretion. Reports come from Europe, Great Britain, Canada and the United States. The paucity of reports from Latin America is interesting. There is no obvious explanation for this.

The onset of the disease is marked by sudden, severe pain. This is usually preceded by vomiting; it may be followed by vomiting or may occur without vomiting. The pain may be substernal, epigastric or abdominal. It is characteristically so severe that large doses of narcotics are required to relieve it. A band-like pain surrounding the body at the level of the diaphragm has been described. Not uncommonly the patient feels a sensation of something having torn or given way. Pleural or shoulder pain is a rather common symptom depending on the presence of pleural contamination and effusion. The pain is made worse by deep breathing, swallowing or by taking anything by mouth. It is improved by the patient assuming a sitting or half-erect position. Following the pain and vomiting are dyspnea, shock and cyanosis. The degree of cyanosis will depend on the severity of the dyspnea, hypoxia and pain. The dyspnea is caused by hydrothorax, pneumothorax and pain. Mediastinal and subcutaneous emphysema may add to the patient's dyspnea and discomfort. The state of shock will depend on the degree of pain and hypoxia, and upon the amount of blood loss.

Severe, sudden pain usually associated with vomiting is nearly always the first symptom. Other symptoms will soon follow and will become increasingly obvious within a few hours. Thirst is an early, common and often extreme symptom. Several writers have noted a change in the quality of the patient's voice. This is thought to be due to the mediastinal emphysema and may precede the appearance of subcutaneous emphysema.

EXAMINATION

Physical Examination. If the patient is seen very soon after the rupture, he will be experiencing severe pain but may not appear severely ill. He will be sitting in a semi-erect position or lying on his side with his knees drawn-up. The pulse is often not greatly elevated, usually 90 to 100. The temperature in the first few hours runs between 99° and 100°F. The respiratory rate is elevated to 30 or 40. Dyspnea, hypoxia, cyanosis and shock are always present to some degree and may rapidly increase. The blood pressure is usually normal. It may become low as the disease progresses or in the presence of unusual blood loss.

TABLE III
PHYSICAL FINDINGS

Findings	Per cent in Series of Priviteri and Gay [20]	Per cent in Fifty Cases Reviewed
Hydrothorax	85	98
Emphysema	62	88
Hydrothorax, left	60	78
Hydrothorax, right	33	10
Hydrothorax, bilateral	7	20
Air beneath diaphragm	0	0

Examination of the skin and subcutaneous tissue shows subcutaneous emphysema in most of the cases. (Table III.) This may or may not be an early finding but usually appears from one hour to several hours after the rupture has occurred. It may be generalized throughout the upper chest and face; however, it is most often noted in the left supraclavicular region, next most often in the right supraclavicular area and then in the suprasternal notch. In one personally observed case subcutaneous emphysema of the neck and face was the presenting complaint. It was not associated with pain or vomiting.

Early in the disease, examination of the chest may be entirely negative. Pleural effusion usually appears within a short time. The typical findings, including lack of breath sounds and dullness over the area of the effusion, can easily be noted. Pleural effusion is most commonly on the left side and may occur on the right side or bilaterally; rarely is it absent. (Table III.)

Rigidity of the abdomen is common, often confined to the upper abdomen. Peristalsis is reduced. Reflexes are hyperactive throughout. The remaining examination is usually non-contributory.

Laboratory Examination. Roentgenographic examination of the chest is the most important laboratory procedure to be carried out in a case of suspected rupture of the esophagus. The x-rays should be taken with the patient in the upright position if possible and also in the lateral decubitis position. They should include the diaphragm, chest and soft tissues of the neck. Common findings are mediastinal emphysema, subcutaneous emphysema, pleural effusion, pneumothorax and widening of the mediastinum. (Fig. 1.)

Free air beneath the diaphragm has never been described in rupture of the esophagus. (Table III.) If this condition is present, one is dealing with perforation of an intra-abdominal organ. The presence of air behind the heart or in the mediastinum is pathognomonic. Presence of air in the tissue usually around the base of the neck is an early sign of subcutaneous emphysema.

Hydropneumothorax in one or both of the pleural cavities is usually present. If the mediastinal pleura ruptures immediately, the contents of the stomach will go directly into the pleural cavity, causing an immediate hydropneumothorax with little or no mediastinal or subcutaneous emphysema. If the mediastinal pleura remains intact, mediastinal and subcutaneous emphysema will be a prominent finding with little or no fluid in the pleural cavity. A fluid level within the mediastinum is diagnostic of a rupture of the esophagus.

If a diagnosis is in doubt, the perforation can often be outlined by introducing lipiodol or barium into the esophagus. (Fig. 1.) If the patient is comatose or moribund, intraesophageal instillation can be accomplished through a Levin tube. The use of lipiodol is preferred; however, some authors have suggested that barium is more likely to outline the perforation. The presence of barium in the pleural cavity is of no significance since thoracotomy is to be carried out immediately.

Other laboratory procedures are useless in early diagnosis. The blood count may show an elevation due to the dehydration or infection. Occasionally hemorrhage will occur with a drop in the blood count. None of these changes will be shown in the first few hours.

DIAGNOSIS

The diagnosis of this condition can often be made by history and physical examination alone. A diagnostic triad has been emphasized by Barrett [3]: (1) rapid respiration, (2) abdominal rigidity and (3) subcutaneous emphysema. A second triad was suggested in Mackler's paper [13], consisting of: (1) vomiting, (2) low thoracic pain and (3) emphysema of the neck. A diagnosis of this condition is usually not difficult if one thinks of it. If the diagnosis is in doubt, x-ray examination of the chest, neck and abdomen is most important. Aspiration of the fluid within the chest cavity usually reveals a dark, turbid fluid containing food particles. If the pH of the material is determined, it will most commonly be acid. Often it will have the odor of gastric contents. If one is still in doubt of the diagnosis or in doubt of the position of the perforation, the perforation should be outlined by lipiodol or barium, as one prefers. This is easily demonstrated. The opaque material will run freely out into the pleural cavity. (Fig. 1.) Occasionally it will remain within the tissues of the mediastinum. If the diagnosis remains in doubt, instillation of the blue or red dye into the esophagus and its recovery from the pleural fluid is sometimes useful to demonstrate an abnormal communication. This procedure is not always accurate, as pointed out by Starkey [23], Naclerio and others.

Differential Diagnosis. Ruptured esophagus is most frequently confused with ruptured peptic ulcer, acute pancreatitis or coronary thrombosis. In addition to these, this condition may simulate mesenteric thrombosis, dissecting aortic aneurysm, pulmonary embolism, spontaneous pneumothorax, rupture of a bronchus, volvulus, intestinal obstruction, strangulated bowel, ruptured gallbladder and diaphragmatic hernia associated with rupture of the stomach or bowel. If the patient relates the usual history, exhibits the diagnostic triads mentioned heretofore, has fluid in the chest and lacks free air under the diaphragm, most of the other confusing conditions can be quickly ruled out. Most diagnostic failures are due to the fact that the examiner is unaware of the symptoms of rupture of the esophagus or simply fails to think of it.

Mesenteric thrombosis, coronary thrombosis, perforated intra-abdominal viscus, intestinal obstruction, strangulated bowel and volvulus seldom if ever cause fluid in the chest and are never the cause of subcutaneous or mediastinal emphysema. Some of these conditions are frequently associated with free air beneath the diaphragm, a finding always absent in esophageal rupture.

FIG. 1. X-ray of a typical patient showing loculated hydropneumothorax with no free air beneath the diaphragm. Subcutaneous emphysema is present. Note lipiodol in the left pleural space, present after a lipiodol swallow.

Rupture of the gallbladder may cause fluid in the chest and is rarely associated with air beneath the diaphragm; however, it is frequently associated with a previous history of gallbladder disease. Often an obvious gallstone is seen in the roentgenogram and it is never associated with subcutaneous emphysema. Acute pancreatitis can usually be ruled out by determining the level of the blood amylase. Acute pancreatitis never causes mediastinal or subcutaneous emphysema but may cause pleural effusion. It is often associated with severe abdominal pain and rigidity, and with shock.

Pulmonary infarction and/or embolus never causes emphysema. They are often associated with cough and hemoptysis which are not seen in rupture of the esophagus. Severe abdominal pain and rigidity are not present as in esophageal rupture.

Spontaneous pneumothorax may produce any of the symptoms found in rupture of the esophagus but as a rule does not cause the abdominal pain or severe shock. There is usually a high degree of collapse of the involved lung, and fluid rarely occurs at the onset but

TABLE IV
RESULTS OF TREATMENT (FIFTY CASES)

Technic	Living	Dead
Thoracotomy and closure of rupture	14	5
Surgical drainage	1	8
No surgical therapy	0	22

makes its appearance in twenty-four to forty-eight hours. Subcutaneous emphysema is rarely seen in this condition. Rupture of the bronchus may cause severe subcutaneous and mediastinal emphysema with pain. There may be a collapse of the lung with severe dyspnea, shock and prostration. Formation of fluid does not occur as an early finding but may occur later. Neither of the last two conditions mentioned is commonly associated with the symptoms of vomiting, retching or other gastrointestinal symptoms. They rarely cause epigastric pain and are never associated with a board-like rigidity of the abdomen.

Rupture of a previously herniated stomach or bowel into the mediastinum with an extension into the pleural cavity may simulate rupture of the esophagus in every respect. Careful fluoroscopic and roentgenographic examination, during which the patient swallows some barium or lipiodol, will often demonstrate the herniated stomach and suggest the diagnosis. Since the treatment of this condition is the same as the treatment for ruptured esophagus, the differentiation of these two conditions is of only didactic importance. In either case one performs an immediate operation with closure of the rupture.

PROGNOSIS

The prognosis of rupture of the esophagus is always extremely grave. In a review of fifty-three cases by Kinsella et al. [12] thirteen patients died in less than twelve hours, twenty-four in less than twenty-four hours and eight in less than forty-eight hours; only eight patients survived over forty-eight hours. Early deaths are due to shock, tension pneumothorax or possibly hemorrhage. Later deaths are due to pleural or mediastinal infection. In 1946 Eliason and Welty [9] reported a mortality of 100 per cent in these cases, with the exception of the two cases reported by Graham. Derbes and Mitchell state that 25 per cent of these patients are dead within twelve hours and only 35 per cent survive the twenty-four hours. Eleven per cent live forty-eight hours and none live over a week without treatment.

Although the prognosis is influenced by the size and the position of the rupture, death is almost certain to occur unless the diagnosis is made and adequate treatment is carried out at the earliest possible time. In these days of plentiful antibiotics, of the judicious use of blood and fluids and of prompt surgical intervention, the patient can be offered a much better prognosis.

TREATMENT

No patient with spontaneous rupture of the esophagus has survived without surgical treatment. Thoracotomy with closure of the perforation offers the best results. Since 25 per cent of these patients are dead within twelve hours, the need for haste in diagnosis and treatment is great. Of fifty cases reviewed, fourteen of nineteen patients survived after thoracotomy and closure, and only one of nine survived after simple drainage. Twenty-two untreated patients died. (Table IV.) Samson [21] reported fifteen cases in which thoracotomy was employed; ten patients survived with fewer complications. Only eight of the fifteen patients treated with drainage survived and these had more complications and a longer hospital stay.

The presence of shock should not delay surgery. The shock in these cases is due to pain, hypoxia and occasionally to blood loss. Tension pneumothorax, pleural fluid and inadequate ventilation result in hypoxia. Under anesthesia the pain stimulus is gone and respiration improves. As the chest is entered, the fluid and air are removed and the lung is expanded. The blood loss is replaced, shock is lessened and the patient does well during the procedure.

Nothing is to be gained by procrastination. The shock is treated, and with proper anesthesia and controlled respiration thoracotomy is performed. The rent in the esophagus is found and closed with two layers of non-absorbable suture, making sure to close the

mucosa carefully. The pleural cavity is cleaned out and the lung re-expanded. Decortication is done if needed. A plastic tube is passed down the esophagus and into the stomach. This is used first for decompression and then for feeding. The mediastinal pleura is opened widely to facilitate drainage. The pleural cavity and mediastinum are d brided and antibiotics are instilled. The chest is then closed, adequately draining the mediastinum, pleural cavity and site of repair.

Some writers believe that the use of intraluminal tubes interferes with the healing of the esophagus. They prefer intravenous feeding. If another method of feeding is desired, jejunostomy should be used. Gastrostomy is often unsatisfactory because of the reflux of the feedings into the esophagus.

If thoracotomy is not feasible or must be delayed, thoracentesis should be carried out, followed as soon as possible with closed suction drainage. Everything by mouth must be stopped. A Levin tube should be placed into the esophagus and down into the stomach for decompression but not for feeding. Feeding should be done parenterally or through a jejunostomy.

Supportive therapy should be started immediately and should be continued during and after any operative procedure. This consists of adequate blood and fluids, oxygen, antibiotics, vitamins and high caloric intake. Narcotics and sedatives should be used to relieve pain without causing undue respiratory depression.

Common postoperative complications are empyema, mediastinitis and breakdown of the repair. To avoid these, adequate cleansing, débridement and drainage of the pleural cavity and mediastinum are imperative. Meticulous closure of the mucosa at the site of rupture is most important to avoid leaks. Muscle and pleura to support the repaired area is desirable but seldom possible. Immediate and complete expansion of the lung is absolutely necessary. Atelectasis should be treated with immediate bronchoscopy. If fistula does occur, local drainage is essential.

Following surgery the esophagus is kept at rest for a week or more and the patient is fed through a Levin tube, parenterally or by jejunostomy. Because of necrosis of the tissue at the site of the rupture, healing is often retarded and thus requires a longer period before feeding by mouth is safe. Just when the patient can be fed by mouth can often be determined by the use of lipiodol and fluoroscopy.

Many patients have survived after simple drainage and one has survived after thoracentesis. However, the mortality and morbidity is much higher in this group. It has been stated that drainage is safe after twenty-four to forty-eight hours. Drainage is satisfactory only after the rupture has closed or has been walled off. In any given case this may occur in a few hours or may never occur. It is more likely to have occurred if the patient is still living after one to several days. If the patient is seen within twenty-four hours of the rupture, thoracotomy and closure should be carried out immediately. The same is true in most instances after twenty-four hours, although drainage is more likely to be successful in that patient who is still living twenty-four hours or longer. Thoracotomy with closure of the rupture remains the procedure of choice in most cases.

SUMMARY

1. So-called spontaneous rupture of the esophagus may be divided into three groups: (1) those due to esophagitis with ulceration, (2) those due to increased intraluminal pressure within the esophagus, and (3) those truly spontaneous cases in which there is no obvious cause.

2. Rupture of this type occurs most commonly at the lower end of the esophagus on the left posterolateral wall of the organ.

3. Most patients give a history of vomiting followed with pain in the chest or upper abdomen, followed with dyspnea, cyanosis and shock.

4. Common physical findings are: pleural effusion, mediastinal and subcutaneous emphysema, and upper abdominal rigidity.

5. Diagnosis is confirmed by chest x-ray, barium or lipiodol study of the esophagus, and study of the pleural fluid.

6. Although patients have survived after surgical drainage, the treatment of choice is immediate thoracotomy with closure of the rupture.

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The "V Sign" in the Diagnosis of Spontaneous Rupture of the Esophagus (An Early Roentgen Clue)

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ONSET of spontaneous rupture of the esophagus is usually sudden and dramatic, and it rapidly leads to a state of shock. Invariably, in a short period of time irreversible toxemia, hypoxemia and peripheral collapse occur, and death almost always follows within twenty-four to forty-eight hours. Unless the condition is diagnosed early and immediate thoracotomy with closure of the perforation is carried out, the patient rarely survives.

Up to approximately ten years ago the mortality rate was 100 per cent. Today the mortality is still high [1]. In many cases the diagnosis is not made and the condition is not even suspected before death. In many of these instances the disease is usually first discovered at autopsy.

The etiology of spontaneous rupture is unknown. Most authorities contend that the tremendous increases in intraesophageal pressure in an apparently normal esophagus, associated with violent vomiting and retching, is responsible for the tear. The esophagus ruptures at its weakest point just above the cardia [1–3]. The tear is usually single and longitudinal, and is located in the lower end of the esophagus. In these cases no disease process, either gross or microscopic, is demonstrable at the site of the rupture.

The diagnosis of spontaneous rupture can often be made with ease if this condition is considered in the differential diagnosis of any thoracic or abdominal emergency. The mistaken diagnosis of ruptured peptic ulcer has been made many times, especially by surgeons, and exploratory laparotomies have been performed (Case 1) [2]. The internist frequently confuses the condition with acute myocardial infarction (Case 11) [1]. Chest physicians may lean toward a diagnosis of ruptured emphysematous bleb if mediastinal and subcutaneous emphysema is found on physical examination and x-ray study (Case 11). Other common errors in diagnosis are acute pancreatitis, mesenteric thrombosis and dissecting aneurysm of the aorta. Spontaneous pneumothorax, pulmonary interstitial emphysema and pulmonary embolism are other diagnoses which are infrequently made.

The most valuable single diagnostic aid is the roentgenogram of the chest. This film will demonstrate mediastinal and/or subcutaneous emphysema, hydrothorax or hydropneumothorax. (Fig. 1A, B, and C.) Most significant and practically diagnostic is the finding of localized mediastinal emphysema, which can easily be overlooked; this is usually evident before physical signs are present. (Figs. 1A, 2 and 5.)

Kinsella, Morse and Hertzog [2] state, "The earliest finding on xray films, probably present in all cases from the onset, if recognized, is the presence of emphysema in the mediastinum, localized at first, later more extensive, involving the whole mediastinum, and eventually fanning out as subcutaneous emphysema in the cervical region."

Ware, Shnider and Davis [3] emphasize the importance of detecting mediastinal emphysema before it has spread. They state, "Early examination of chest xrays for evidence of air in the mediastinum or retrocardiac area is mandatory, since its presence may antedate subcutaneous emphysema by a number of hours." (Cases 1 and 11.) The authors add that

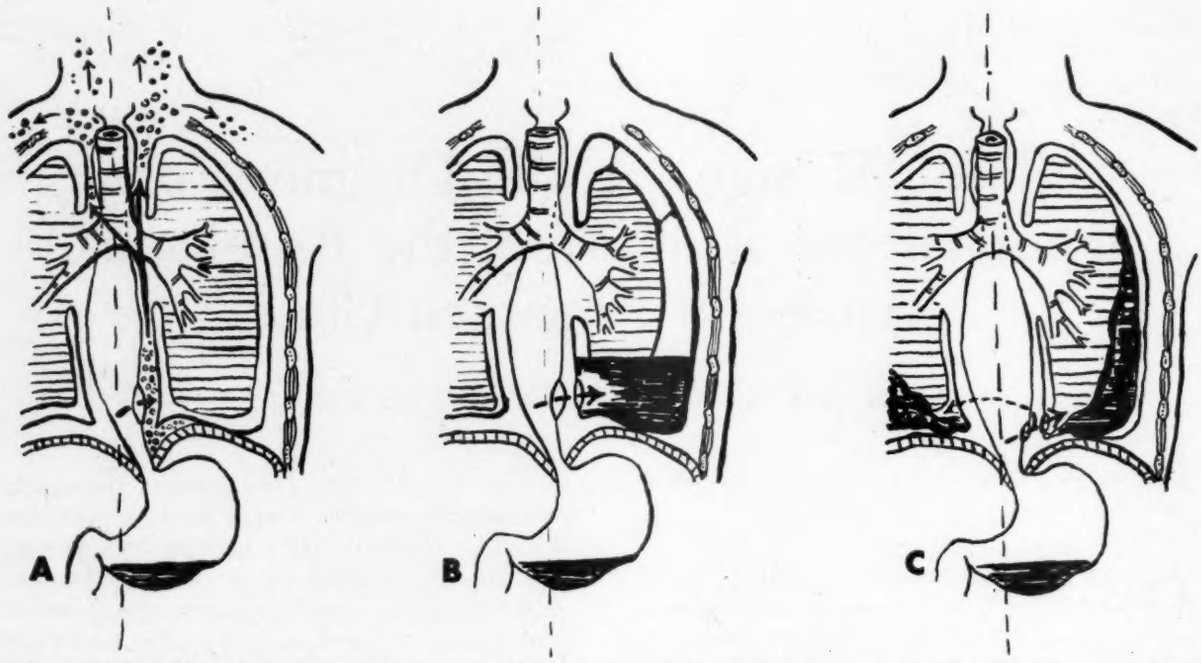

FIG. 1. A, localized mediastinal emphysema. Following rupture if the pleura remains intact, the air and/or fluid remains localized in the mediastinum and local mediastinal emphysema results. The air then travels up through the mediastinum into the subcutaneous tissues of the neck and face. B, left hydropneumothorax. If adhesions exist between the esophagus and pleura at the site of rupture, perforation will occur directly into the pleural cavity, giving rise to hydropneumothorax; when this develops early, subcutaneous emphysema appears late or not at all. C, bilateral hydrothorax. With a localized mediastinal process, as a result of mediastinitis, left-sided pleural effusion rapidly develops; in a number of instances it is bilateral.

if emphysema is to occur, it will be noted first in the mediastinum.

Lynch [4] states, "Since most patients are hospitalized almost immediately because of the severe nature of their illness, and since perforated ulcer is suspected, it is usual to have an xray examination of the abdomen in a search for subphrenic air. Such a procedure can be most helpful in the diagnosis, since air behind the heart is frequently seen and is one of the most characteristic findings. Even in the early stages, a small pleural effusion has been noted. The usual chest xray film at this time may also show air in the upper mediastinum. Under the circumstances here described, such a finding is diagnostic."

In view of the paucity of reported cases in which x-ray films reveal localized mediastinal emphysema, it seemed of value to report two such cases. In each case the linear roentgen density corresponded to the fascial planes of the mediastinal and diaphragmatic pleurae in the region of the lower esophagus. (Figs. 1A, 2 and 5.) Since the roentgen configuration takes the form of the letter V, it may be conveniently referred to as the "V sign."

The following cases clearly reveal the clinical picture which is so characteristic of spontaneous rupture of the esophagus and also bring to light problems that arise in its management.

CASE REPORTS

CASE 1. D. M., a fifty-five year old white man, was admitted to Columbus Hospital at 4 P.M. on April 9, 1954. He stated that following his noon meal he had become nauseous and then had vomited and retched violently. While retching he was seized with severe colicky pain in the lower sternal and mid-epigastric regions. The intern's admission workup revealed that the patient's blood pressure was 128/74, pulse 120, respirations 32 and temperature 100.2°F.

Fifteen minutes after the time of admission the patient was seen by an attending physician on the medical service, whose note reads as follows: "Patient seen a few minutes after admission. He was doubled up because of severe abdominal pain and because of the pain, he was not cooperative. Patient stated that about 1 P.M. he had become nauseous and vomited three times. His brother told me that the patient was treated for ulcers six years ago.

In spite of the injection of morphine sulfate given where he works, and 100 mg. of demerol, his pain persisted. Heart sounds of fair quality, rate and rhythm regular but rapid. No murmurs. Lungs—negative to percussion and auscultation. Abdomen—marked tenderness in the right upper quadrant and patient points with one finger to a definite point of tenderness. Abdomen is very rigid with rebound tenderness. Impression—ruptured viscus."

At 5 P.M. on surgical consultation it was learned that the patient had a questionable prolapse of gastric mucosa into the duodenum and a possible duodenal ulcer, for which he was being treated.

When the patient was first seen by the examiners he was sitting in bed and grunting; he had definite pallor. Abdominal examination demonstrated marked rigidity of both rectus muscles, with greatest degree of spasticity in the upper abdomen; there was a maximum point of tenderness over the duodenal area. The tentative diagnosis was ruptured duodenal ulcer.

In order to corroborate the diagnosis an erect posteroanterior film of the upper abdomen was requested to detect the presence of subphrenic air. (Fig. 2.) The red blood count was 4,800,000; the white blood count was 18,000, with 90 per cent polymorphonuclear leukocytes.

At 6:45 P.M. laparotomy was performed under spinal anesthesia and complete exploration of the abdominal cavity revealed no abnormalities. At the close of the operation the surgeon stated that the patient should have continuous gastric suction and that the site of possible pathologic disease should be sought elsewhere.

On April 10th at 11:00 A.M. the patient appeared to be more comfortable. His abdomen was less tender. Examination of the chest revealed no rales but there was diminished resonance in the left base, posteriorly.

At 4:30 P.M. the patient had labored breathing. There were numerous crepitant and subcrepitant rales in the left base, posteriorly. The impression at this time was that a pneumonic process was developing.

On April 11th the patient was more comfortable but still had labored breathing. There was evidence of subcutaneous emphysema in the supra- and infraclavicular regions. The diagnosis of a ruptured emphysematous bleb of the lung was considered. Auscultation of the chest revealed crepitant and sub-crepitant rales at the left base. There was dullness on percussion over the same area. An x-ray of the chest demonstrated a shadow suggestive of pneumonitis of the left lower lobe and left pleural effusion.

FIG. 2. Case 1. Patient D. M. Admission film (patient in erect position). Note the localized mediastinal emphysema. The linear density corresponds to the fascial planes of the mediastinal and diaphragmatic pleurae in the region of the lower esophagus. This is easily overlooked; it is usually evident before physical signs are present.

On April 12th the patient was more comfortable. His respirations were 42, temperature 100.6°F., and pulse 112. A thoracic surgical consultation was requested.

At 2:30 P.M., at the thoracic surgical consultation, the author strongly considered the diagnosis of spontaneous rupture of the esophagus and requested an immediate lipiodol swallow. Extravasation of the oil into the mediastinum was revealed. (Fig. 3.) Despite this extravasation the radiologist was hesitant about the diagnosis of spontaneous rupture of the esophagus. He leaned toward a diagnosis of diverticulum of the distal esophagus. In order to confirm the diagnosis further, the author suggested that if the patient were made to swallow methylene blue, its recovery from the pleural cavity by thoracentesis would prove the diagnosis. On thoracentesis 460 cc. of straw-colored fluid were removed from the left pleural cavity but the fluid did not contain any dye. However, in view of the clinical course and the presence of extravasated lipiodol in

FIG. 3. Case 1. X-ray of chest taken on fourth hospital day, just prior to thoracotomy. Note the presence of bilateral effusion, mediastinal and subcutaneous emphysema and the extravasation of lipiodol into the mediastinum.

the mediastinum, the patient was immediately subjected to a thoracotomy.

At operation at 4:15 P.M., under local anesthesia, 1 per cent novocain, paravertebral intercostal block, a long segment of the left ninth rib was resected. The pleural cavity was entered and numerous loculations containing serosanguineous fluid mixed with brownish, fibrinoexudative material were encountered. Again there was no evidence of methylene blue. The pleural linings were fiery red. The lung was collapsed approximately 20 per cent and it was adhered to the diaphragm and mediastinal structures. The pleural space was evacuated of its contents and was irrigated with 3 quarts of saline solution. The actual site of rupture in the esophagus was not delineated, but in the region of the perforation a fibrinous pocket was present. When this pocket was perforated, a moderate amount of straw-colored fluid, lipiodol and methylene blue dye was encountered. (Fig. 4.) The esophagus was markedly congested, and an abundance of loose, necrotic tissue was adhered to it. The mediastinal

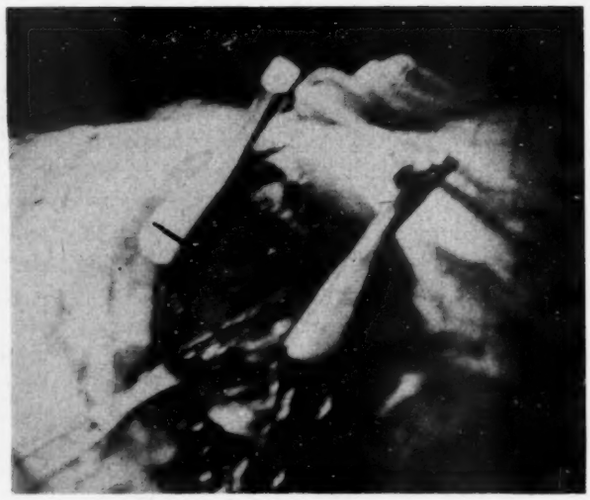

FIG. 4. Case 1. Operation demonstrating an indentation in a fibrinous wall. This comprised a periesophageal loculation. In it was contained the methylene blue which was ingested and could not be recovered by thoracentesis. If a physician relies on its recovery as a specific diagnostic test, he might be led astray.

pleura was opened widely. The chest cavity was then drained with gauze packing and rubber Penrose sheaths.

Postoperatively the nasogastric suction, which was started at the time of the laparotomy, was continued for several days and the patient was given intravenous blood, plasma and electrolyte fluids. The nasogastric tube seemed to be inadequate for decompressing the stomach.

On April 19th a Stamm gastrostomy was performed under local novocain field block for decompression and alimentation purposes. During the first week after thoracotomy, right-sided pleural effusion continued to develop despite adequate drainage of the mediastinal and pleural cavities. The effusion was removed by repeated thoracenteses.

On April 20th the potassium and sodium levels were 2.9 and 146.7 mEq./L., respectively. Substitution therapy was instituted.

On April 23rd a Witzel jejunostomy was performed under local anesthesia at a distance of approximately 8 inches from the ligament of Treitz.

The chest drained adequately. The following therapy was instituted: broad-spectrum drugs, blood and electrolyte fluids. However, the patient's course continued to go downhill and he died at 7 P.M. on April 27th, fifteen days after the thoracotomy.

Comment. This case illustrates the typical

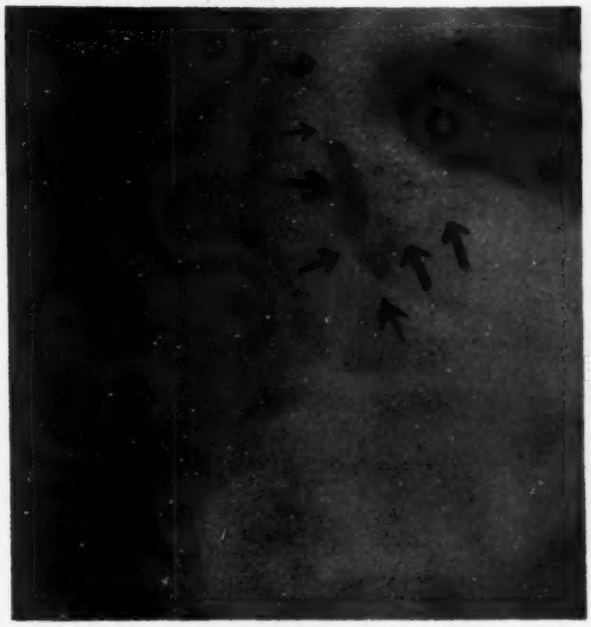

FIG. 5. Case II. Patient G. M. Admission film demonstrates (as in Figure 2, Case 1) local mediastinal emphysema in the lower mediastinum.

FIG. 6. Case II. X-ray of chest showing mediastinal and subcutaneous emphysema. There is also bilateral emphysema of both lungs, with flattening of the right hemidiaphragm. The left lower lobe region is suggestive of pneumonitis or hydrothorax. X-ray patterns do simulate other disease entities. Therefore, unless the diagnosis of spontaneous rupture of the esophagus is thought of, it cannot be made.

clinical picture and course of esophageal rupture. In spite of this the diagnosis of the first observers was ruptured duodenal ulcer rather than esophageal rupture. The x-ray taken for the presence of subphrenic air soon after the patient's admission to the hospital clearly demonstrated localized mediastinal emphysema in the lower mediastinum; this finding should have aroused suspicion of the esophageal rupture.

CASE II. G. M., a sixty-two year old white man, was examined in the Harlem Hospital accident room at 12:20 A.M. on April 24, 1953. In view of the patient's history of having had great difficulty in breathing, severe pain in the substernal region and rales in both lungs, the admitting physician made a diagnosis of acute myocardial infarction.

The history taken by the medical intern soon after admission revealed that the patient's illness dated back to six days before admission, when immediately after supper, after drinking several bottles of beer, the patient had vomited and retched for approximately half an hour. Since that time he had been nauseous, had belched a great deal and had passed large quantities of flatus. He was not able to keep any food down. Approximately five hours before admission the patient again began to retch and was suddenly seized with severe pain in the substernal and epigastric regions. Because of the pain, which was continuous and was becoming progressively worse, and because of inability to catch his breath, the patient was rushed to the hospital.

When examined the patient was sitting up on the stretcher with flexion of the knees to the abdomen and was most apprehensive. He also had rapid and shallow breathing and was cyanotic. At that time his blood pressure was 78/50, pulse 110 and respirations 38. There was marked distention of the cervical veins. Subcutaneous emphysema in the left supraclavicular region could be seen and palpation revealed crepitation. The chest was barrel-shaped. Percussion revealed hyper-resonance over both lung fields. The heart tones were distant and apparently negative. Varicose veins were present in both legs. Tentative diagnoses by the intern were: (1) possible ruptured viscus, (2) acute intestinal obstruction, (3) ruptured aneurysm of the descending aorta, or (4) chronic pulmonary emphysema. He ordered immediate oxygen therapy, 500 cc. of plasma,

FIG. 7A. Case 11. X-ray taken immediately following lipiodol swallow. Since the patient was seriously ill, most of the oil entered the tracheobronchial tree. This may arouse suspicion of a tracheoesophageal fistula due to a malignant lesion, as it did in this case. It may lead a physician astray or may cause delay in arriving at the correct diagnosis. This can be prevented. (See Figure 7B.)

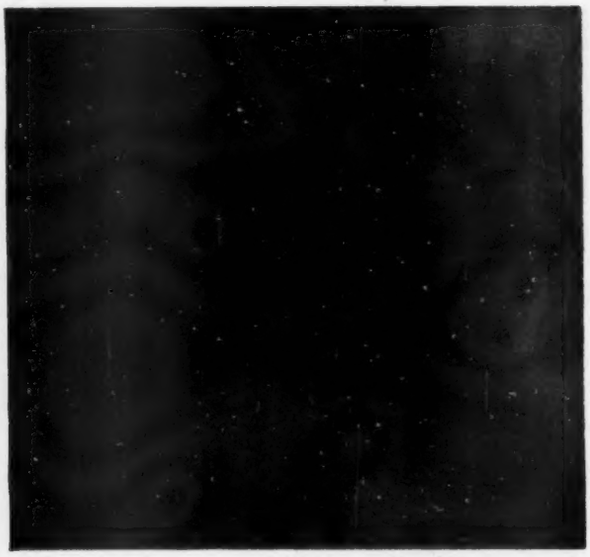

FIG. 7B. Case 11. X-ray demonstrating extravasation of oil into the mediastinum. The oil was instilled into the esophagus through a Levin tube.

blood for typing and cross matching, emergency x-ray of the chest, posteroanterior view, and a flat plate upright film of the abdomen. (Fig. 5.)

At 7 A.M. the medical resident examined the patient and reviewed the x-rays. He made a diagnosis of subcutaneous emphysema due to a ruptured emphysematous bleb.

At 8:30 A.M. the medical chest resident made a diagnosis of spontaneous pneumothorax and subcutaneous emphysema.

At 11 A.M. the members of the chest medical service, while making rounds, stopped to see the patient. After examining him and carefully studying the x-rays (Figs. 5 and 6), they made a diagnosis of: (1) bronchogenic carcinoma with pleural rupture, mediastinal and subcutaneous emphysema, and (2) chronic pulmonary emphysema and fibrosis. They requested an immediate thoracic surgical consultation.

At 1:30 P.M., at the thoracic surgical consultation, the patient was desperately ill, cyanotic and moribund. He manifested signs of mechanical compression of the vascular structures in the mediastinum. A diagnosis of spontaneous rupture of the esophagus was immediately considered. The patient was asked to swallow approximately 10 cc. of lipiodol; the oil was poorly visualized in the periesophageal area but was clearly seen in the left main bronchus. (Fig. 7A.) Since this test was not sufficiently diagnostic to prompt immediate thoracotomy, the author requested that a Levin tube be passed into the esophagus and that lipiodol be injected through it. Repeat x-rays of the chest clearly demonstrated the extravasated oil in the mediastinum and the presence of an esophageal fistula. (Fig. 7B.)

At operation at 4:15 P.M. on April 24th the left pleural cavity was entered through the bed of the ninth rib. Approximately 1,000 cc. of brownish, turbid, foul-smelling liquid material, containing what appeared to be food particles and plastic exudate, were encountered. The lung was markedly emphysematous and adhered to the parietes. The parietal and visceral pleural linings were congested and covered with plastic exudate. The mediastinal pleura was bulging and tense, and prevented spillage of mediastinal contents into the pleural cavity. In the region of the lower third of the esophagus, the mediastinal cavity contained liquid material, exudate and lipiodol. The wall of the esophagus was edematous and appeared gangrenous. Approximately 3 fingerbreadths above the diaphragm lipiodol poured out from what seemed to be an esophageal perforation; this region was aspirated of its contents and irrigated with saline. The mediastinal pleura was widely opened. Following this the blood pres-

sure rose and the patient's general condition became slightly improved. The pleural cavity was then packed loosely with gauze dressing and rubber Penrose sheaths. The patient left the operating room in critical condition but slightly improved.

Postoperatively the patient received nasogastric suction, blood transfusions, and adequate fluid and electrolyte therapy. He continued to improve for a short period of time but later went into a state of irreversible shock. His course continued to go downhill and he died on April 26th at 5 P.M., approximately forty-eight hours after thoracotomy. Examination of fluid removed from the chest during surgery revealed the presence of bile.

At autopsy there was no evidence of disruption of the mucosa or wall of the esophagus, except for some roughness of the mucosa with slight loss of the epithelium above the cardia. There was marked congestion of the veins throughout the esophageal mucosa. The tissues between the esophagus and aorta presented a necrotic appearance although the source of the necrosis could not be established; the wall of the aorta was intact throughout. Both lungs were markedly emphysematous and there were bullae in both apexes; numerous adhesions were found between both lungs and the parietes. Other findings were essentially non-significant.

The gross pathologic diagnosis was ruptured esophagus, clinical; no evidence of rupture in pathologic findings (Fig. 8); dilatation of esophageal veins; slight erosion of epithelium in lower esophagus; periesophageal mediastinitis; fibrinous pleuritis, right interlobar; hemorrhagic pulmonary infarction, right lower lobe; passive congestion of lungs, trachea, bronchi, hilar lymph nodes and liver; septic splenitis, accessory spleen.

Comment. This case history again is typical. The diagnoses considered are those which are made so frequently. This case is similar to Case I in that x-rays taken soon after the patient's admission to the hospital showed local mediastinal emphysema in the lower mediastinum; this, as in the other case, also should have aroused suspicion of the esophageal rupture.

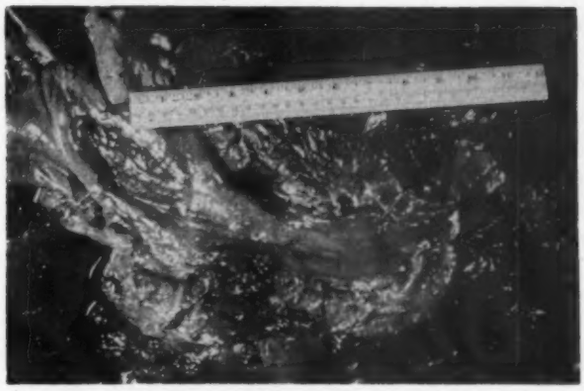

FIG. 8. Case II. Postmortem specimen of esophagus and cardia of stomach. The actual perforation in the esophageal wall could not be identified. A review of the literature reveals that this situation existed in two other instances [7,8].

REMARKS

As shown by these cases and as repeatedly pointed out by previous authors, successful treatment of spontaneous rupture of the esophagus depends on early diagnosis. In the author's cases the postoperative course was rapidly downhill despite the utilization of drainage procedures, modern chemotherapy and supportive measures.

Since early diagnosis gives the optimum chance for survival, it is stressed that the condition be considered in the differential diagnosis of any acute abdominal or thoracic catastrophe, especially in cases in which severe high epigastric or substernal pain occurs during an attack of violent vomiting or retching. Limitation of respiratory movements is usually a leading complaint; this was outstanding in Case II.

The principal diagnostic clinical sign and one which is perhaps most commonly present is subcutaneous emphysema, often first noted in the left supraclavicular area. In Case I subcutaneous emphysema was first clinically manifested in the supra- and infraclavicular regions two days after admission to the hospital; in Case II no mention was made of cervical emphysema by the first observer, but its presence was easily detected by the subsequent examiners.

In a significant number of cases examination of the early x-rays shows localized mediastinal emphysema in the lower mediastinum. This, together with the characteristic history, is practically diagnostic. If this roentgen sign had aroused suspicion of esophageal rupture when the cases presented herein were first observed, the diagnosis could have been easily confirmed. Immediate surgery could have been performed at this most propitious time, i.e., before there was perforation of the pleura; this

would have most likely resulted in cures with a minimum of morbidity. Gardner [5] and Samson [6] each reported a case in which the condition was detected and the patient operated upon before perforation of the pleura ensued; the end results were rapid cures. The diagnosis may be established by the demonstration that a swallow of lipiodol extravasates into the mediastinum or pleural cavity; this finding almost always confirms the diagnosis. This procedure revealed extravasation of the oil into the mediastinum in each of four cases of so-called spontaneous rupture [10] and in each of three cases of traumatic rupture of the esophagus treated by the author. Similarly, Overstreet and Ochsner [9] found that a swallow of lipiodol extravasated into the periesophageal tissues in each of eleven cases in which this procedure was used.

Once the diagnosis of spontaneous rupture of the esophagus has been established, treatment should be aimed at wide excision of the mediastinum, direct repair of the site of rupture, expansion of the lung and drainage of the pleural cavity. This operation should be performed despite the shock-like picture that so frequently exists.

SUMMARY

1. X-ray films in two cases, both showing localized mediastinal emphysema in the lower mediastinum, are presented herein. This roentgen sign, together with the characteristic history, should be diagnostic.

2. The cases clearly reveal the clinical picture which is so characteristic of spontaneous rupture of the esophagus and also bring to light problems which arise in its management.

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Cardiospasm (Achalasia of the Cardia)

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THE experience at the Mayo Clinic with the condition known as cardiospasm has been reviewed in a number of communications [*1–3*]. The most recent statistical reports from the Clinic were published in 1951 [*4*] and 1953 [*5,6*], when a series of 601 cases seen in the twelve-year period ending in 1946 were studied. Further statistical analyses will not be attempted in this paper. Rather, our present concepts of diagnosis will be reviewed in the light of recent physiologic observations and methods of treatment will be outlined, with due consideration for our increasing surgical experience in the management of the condition.

Cardiospasm is a term that has been used for many years [*7,8*] for the condition of benign stenosis of the terminal portion of the esophagus associated with deficient or absent peristalsis of the esophageal musculature. Originally the condition was thought to represent spasm at the cardia and hence was called cardiospasm. Later, Hurst and Rake [*9*] noted degeneration of Auerbach's plexus in the esophageal wall and stated that the muscular contractions of the esophagus were inadequate, often failing to reach the cardia. Hurst offered the name achalasia, which means failure to relax, and this term is the best yet proposed to describe the nature of the disturbance in question. Mega-esophagus merely indicates the dilatation of the organ noted in the later stages of the disease and is synonymous with such designations as esophagectasia and dolicho-esophagus.

Ample evidence exists that a physiologic sphincter is present in the terminal part of the esophagus. Like all sphincters, the one just above the esophagogastric junction opens in response to orderly peristaltic movements of the viscus proximal to it. In turn, normal peristalsis apparently depends on the integrity of the tenth cranial nerve and its various ramifications. The exact role of the parasympathetic and sympathetic nerves is not clearly understood, yet disturbances in neurogenic mechanisms of the esophagus do lead to abnormalities in the propagation of peristaltic waves and thus to various types of dysphagia.

Achalasia (or cardiospasm) denotes a state of hypomotility of the esophagus and must be carefully distinguished from the functional disturbance known as diffuse spasm of the esophagus and from localized spasm of the esophagus or cardia. In these latter disorders there is not only hypermotility but also a disturbance in orderly propagation of peristaltic waves. Physiologic methods of recording esophageal motility are now available [*10–12*] and are extremely valuable aids in the differential diagnosis of both functional and organic disorders.

ETIOLOGY

The cause of achalasia is not known. That the condition is a neuromuscular disturbance has been confirmed by both pathologic studies [*9,13*] and physiologic technics. However, no one has explained satisfactorily why degeneration of the ganglionic cells in Auerbach's plexus takes place. In the days when spasm at the

cardia was the prevalent theory, the degenerative changes were thought to be secondary to dilatation and stretching of the walls of the esophagus. However, degenerative changes have been noted in extremely early stages of the disease [*14*]. Etzel [*15*] considered that dietary deficiency of thiamine hydrochloride was responsible for this condition as well as for megacolon (Hirschsprung's disease) and megaureter. Etzel emphasized the poor economic status of Brazilian patients who had cardiospasm, yet in North America authorities agree that all classes of people may be afflicted and that achalasia is not related to either eating habits or financial circumstances.

The contention that achalasia is a psychosomatic disorder has been most controversial [*16*]. It is difficult to refute the argument that the initial malfunction might be related to emotional stress or nervous tension. When the disorder can be recognized clinically by radiologic means, pressure studies or histologic demonstration, the patient is no longer susceptible to psychiatric treatment. Furthermore, in our experience, patients who have cardiospasm are no more neurotic than are any other group of patients.

CLINICAL PICTURE

The principal symptoms of cardiospasm are related to esophageal obstruction. Food sticks in the lower part of the esophagus before it enters the stomach. At times the sensation of obstruction may be referred to the upper portion of the esophagus. Dysphagia may be intermittent in early stages and is often related to the type of food taken. As a rule, warm foods pass more readily than do cold foods and many times the patient will prefer solids to liquids. Regurgitation is common and may be first noted as soiling of the pillow slip. Later, willful regurgitation may be employed to relieve distress.

Inasmuch as esophageal contractions no longer open the cardia, the patient may rely on gravity and hydrostatic pressure to force food from the esophagus to the stomach. At times he will engage in certain exercises and maneuvers to alter the intrathoracic pressure. Thus it is not uncommon in advanced stages of the disease to see the patient with cardiospasm stand up while eating, lean forward with his hands on the mantelpiece or a similar support, grunt and strain with the breath held and glottis closed and finally force food into the stomach.

Pain is not a common symptom of cardiospasm, occurring in less than 30 per cent of our patients [*5*]. It is more prominent in early stages and it is usually localized to the lower substernal region and related to the effort of getting food into the stomach.

The patient who has cardiospasm usually loses weight, but extremely obese patients are seen occasionally. Obvious deficiencies of vitamins are uncommon. Cough is not uncommon, and about 10 per cent of our patients have had demonstrable evidence of aspiration pneumonitis or other complications [*6*]. Bleeding from the esophagus is rare.

ROENTGENOLOGIC FINDINGS

A detailed description of the roentgenologic findings in achalasia has been reported elsewhere [*5*]; a brief summary will be given herein.

In the early stages of cardiospasm, ineffectual hyperactivity may be noted. The primary peristaltic wave is arrested in the upper part of the esophagus and does not travel to the cardia [*17*]. In fact, barium may be "milked" upward instead of passing through the cardia. After some churning about, some of the barium passes into the stomach. In more advanced lesions, the column of barium presents a smooth conical appearance with a beak-like extension into the region of stenosis at the cardia. (Fig. 1.) As the esophagus becomes diffusely dilated the organ becomes passive and motility is absent. In advanced stages, the esophagus may become hugely dilated, tortuous, elongated and saccular.

From a roentgenologic point of view, early cardiospasm may be considered as a "compensated stage" in which frequent, inefficient, poorly co-ordinated esophageal contractions finally force food into the stomach. On the other hand, the esophagus in advanced lesions may be "silent" or in the "decompensated phase" in which only gravity and changes in intrathoracic pressure can overcome the resistance at the cardia.

Pulmonary changes secondary to achalasia already have been mentioned. Most of these are caused by aspiration of retained material and present the picture of aspiration pneumonitis or fibrosis. The shadows noted on the thoracic roentgenogram usually involve the

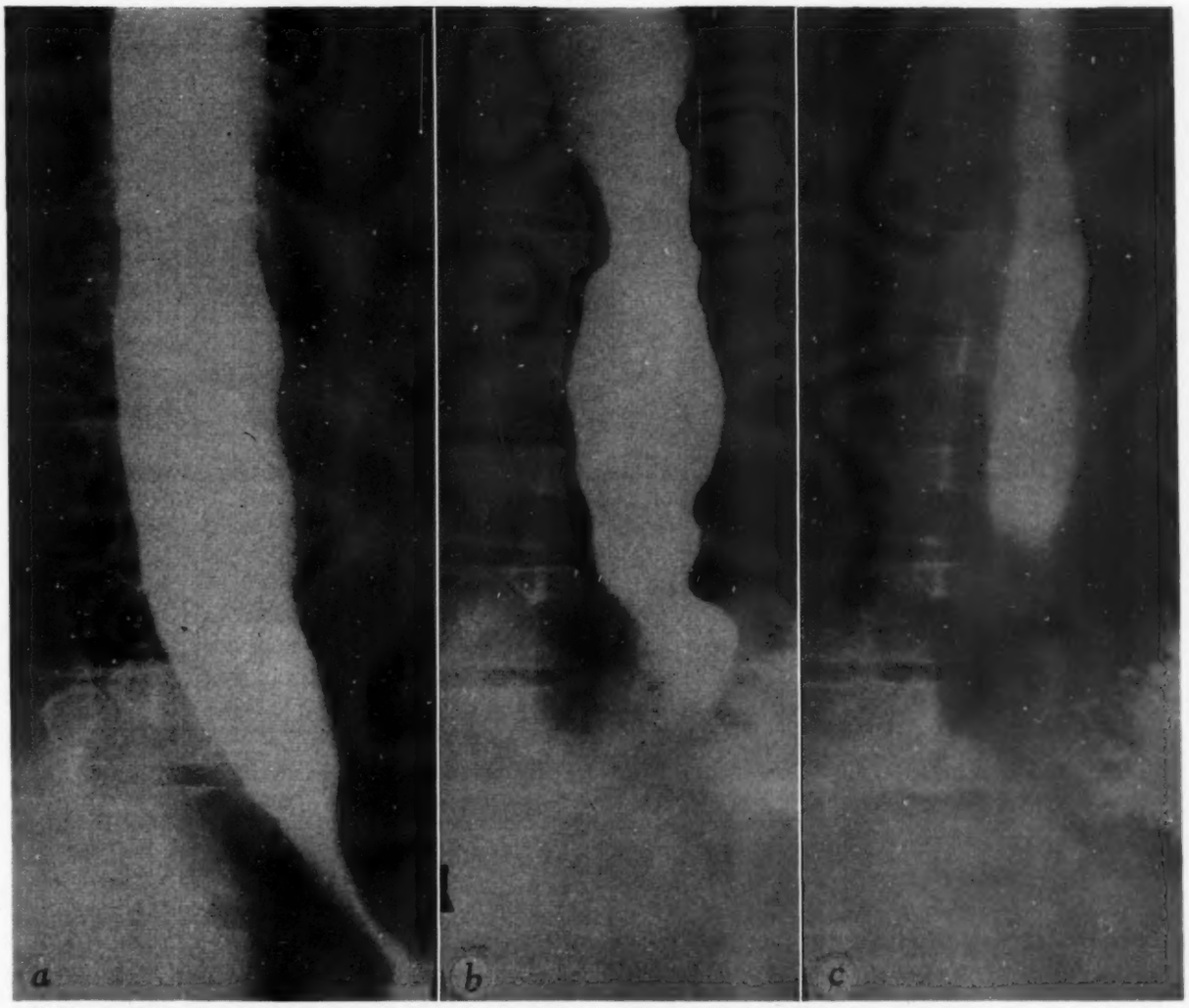

FIG. 1. Roentgenographic appearance of achalasia. (*a*) Characteristic appearance of moderately advanced achalasia. (*b*) Same case; appearance after dilation. (*c*) Same case; appearance after subcutaneous injection of 10 mg. of mecholyl.

mid-portion of the lung fields, especially on the right side.

The dilated esophagus of cardiospasm may produce mediastinal widening on the routine roentgenogram. As a rule, the patient's symptoms will provide an indication for roentgenologic studies after a swallow of barium and thus will lead to the correct diagnosis. If dysphagia is not a prominent symptom and the esophagus is not suspected, mediastinal widening occasionally may pose a diagnostic problem.

PHYSIOLOGIC OBSERVATIONS

Studies of esophageal motility in healthy people and in patients who have cardiospasm have been reported in detail [*10–12,18*]. Measurements of esophageal pressure at rest and during the act of swallowing are made by passing open-tip polyethylene tubes attached to small electromagnetic pressure transducers that are external to the patient. Simultaneous recording of pressures at various levels [*12,13*] permits graphic demonstration of the orderly progression of peristaltic waves in the normal person, whereas in patients who have cardiospasm, peristaltic waves are absent throughout most of the esophagus. (Fig. 2A and B.) In the early stages of cardiospasm, spontaneous changes in pressure often occur, apparently unrelated to the act of swallowing and probably corresponding to the poorly co-ordinated non-peristaltic contractions of the esophagus

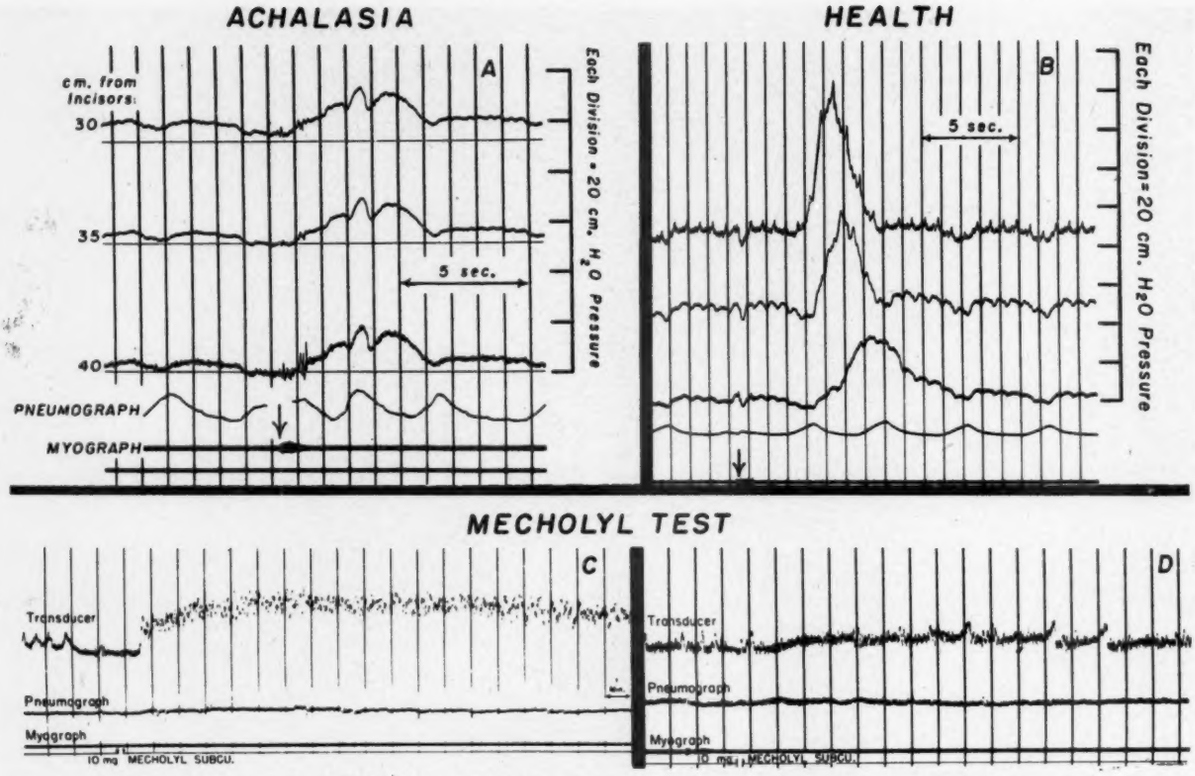

FIG. 2. Esophageal pressures illustrating the essential differences between achalasia and health. A, recording from lower half of esophagus of patient with achalasia, showing absence of peristaltic wave on swallowing. B, normal peristaltic wave recorded from healthy person. C, increase in pressure after subcutaneous injection of 10 mg. of mecholyl in patient with achalasia. D, absence of increase of pressure after injection of 10 mg. of mecholyl in healthy person.

noted roentgenologically. In the more advanced stages, however, motility is practically absent.

Of particular diagnostic significance is the response of achalasia to the injection of methacholine (mecholyl®) chloride [*19,20*]. The patient usually complains of severe substernal pain. The radiologist may see pronounced contraction of the entire esophagus (Fig. 1*b* and *c*), and pressure detectors in place at the time of the injection will show a distinct and prolonged increase in pressure. (Fig. 2C.) In normal persons and in patients who have other esophageal disorders, this response to mecholyl is virtually absent. (Fig. 2D.)

ENDOSCOPIC DIAGNOSIS

Esophagoscopy is not of much value in the diagnosis of cardiospasm but may be important in the differentiation of cardiospasm from other diseases. We do not routinely use esophagoscopy in cases of typical cardiospasm. This examination discloses reddening and thickening of the esophageal mucosa, plus varying amounts of mucus and retained secretions. The stenotic zone at the cardia may give way to the esophagoscope or, in some cases, it may be impossible to pass the instrument into the stomach. Rarely, it becomes necessary to perform esophagoscopy for the purpose of initiating dilation therapy.

The passage of an olive-tipped bougie over a previously swallowed thread is of considerable value in the diagnosis of cardiospasm. In experienced hands the type of resistance noted when the sound passes through the stenotic cardia is unmistakable. Although the obstruction may be firm, it suddenly "gives" and the sensation is different from that experienced with an organic lesion.

DIFFERENTIAL DIAGNOSIS

For obvious reasons it is most important that cardiospasm be differentiated from organic lesions affecting the lower portion of the esophagus, particularly malignant tumors. When an older person with a short history of dysphagia

presents himself, the problem may be difficult. The roentgenologist has much more trouble with the diagnosis of carcinoma of the cardiac end of the stomach than he does with esophageal carcinoma. Gastric carcinomas often encircle the terminal part of the esophagus without invading the mucosa. The roentgenologist may be unable to distinguish the obstruction from that seen in cardiospasm. Likewise, the esophagoscopist may be unable to see a carcinoma of the cardia and biopsy may be unsatisfactory. In such cases, smears and secretions studied by cytologic technics may reveal the true nature of the condition [*21*]. The passage of a No. 41 olive-tipped bougie over a previously swallowed thread may give considerable information concerning the nature of the obstruction. In case of serious doubt, transthoracic exploration of the cardia is indicated.

Benign tumors of the esophagus are rare but large pedunculated fibrolipomas occasionally may give a roentgenologic picture strongly suggesting cardiospasm [*22*]. Even the endoscopist may not recognize the nature of such a lesion, as the mucosa covering such tumors is identical to that of the esophageal wall. Huge sausage-like tumors are particularly likely to give trouble in diagnosis.

The most common benign organic stricture of the lower part of the esophagus is that often associated with hiatal hernias and esophagitis. The patient usually gives a long history of heartburn and "acid indigestion," suggesting an incompetent cardia. By the time the stricture has formed, the esophagus is shortened. In such cases the examiner must be on the lookout for carcinoma in the intrathoracic portion of the stomach.

Scleroderma of the esophagus may present a clinical picture resembling that of cardiospasm, and the roentgenographic changes also may be somewhat similar. The esophageal disorder occasionally will precede the dermatologic manifestations. Raynaud's phenomena are almost invariably present when the esophagus is involved. In many cases of advanced scleroderma one finds a short esophagus with an intrathoracic stomach and a stricture at the esophagogastric junction.

Of the non-organic disorders, diffuse spasm of the esophagus most frequently is confused with achalasia [*23*]. (Table 1.) Diffuse spasm has been described variously as non-sphincteric spasm, pseudodiverticulosis and segmental spasm of the esophagus. It must be distinguished from the asymptomatic corkscrew (curling) esophagus and the "tertiary" contractions sometimes seen roentgenographically in older people, and also from the bizarre irregularity of the esophagus in treated cardio-

TABLE 1
DIFFERENTIAL DIAGNOSIS OF CARDIOSPASM AND DIFFUSE SPASM

Symptoms and Signs	Cardiospasm	Diffuse Spasm
Pain	Uncommon	Almost constant
Obstruction	Always	Variable
Regurgitation	Common	Rare
Retention	Frequent	Never
Psyche	Stable	Nervous
Roentgenologic findings	Diffuse dilatation	Segmental spasm

spasm. (Fig. 1*b*.) This condition tends to occur in nervous, tense people of both sexes but is somewhat more common in women. The most striking symptom is pain, usually substernal and often referred to the back, neck, jaws, ears or even the arms. The pain may or may not be related to eating. Most of these patients have some difficulty in swallowing. The dysphagia is extremely variable and is subject to nervous and emotional influences. Roentgenographically, the condition involves the lower half to two-thirds of the esophagus. It may appear as an irregular spasm (Fig. 3*a*), as diffuse narrowing (Fig. 3*b*) or as multiple pseudodiverticula. (Fig. 3*c*.) Physiologically, as measured by studies of esophageal motility, diffuse spasm is the opposite of cardiospasm or achalasia [*24*]. It is a condition of hypermotility with high pressures, repetitive waves and simultaneous contractions at various levels in response to the stimulus of swallowing. Diffuse spasm frequently is confused with early cardiospasm, both clinically and roentgenoscopically. Measurements of pressure and the mecholyl test are extremely helpful in making the differentiation. The findings are different when bougies are passed and, of course, diffuse spasm responds less well to treatment than does cardiospasm. Diffuse spasm frequently is associated with some other lesion of the digestive tract such as duodenal ulcer, cholelithiasis, pancreatitis or hiatal hernia [*25*]. The correction

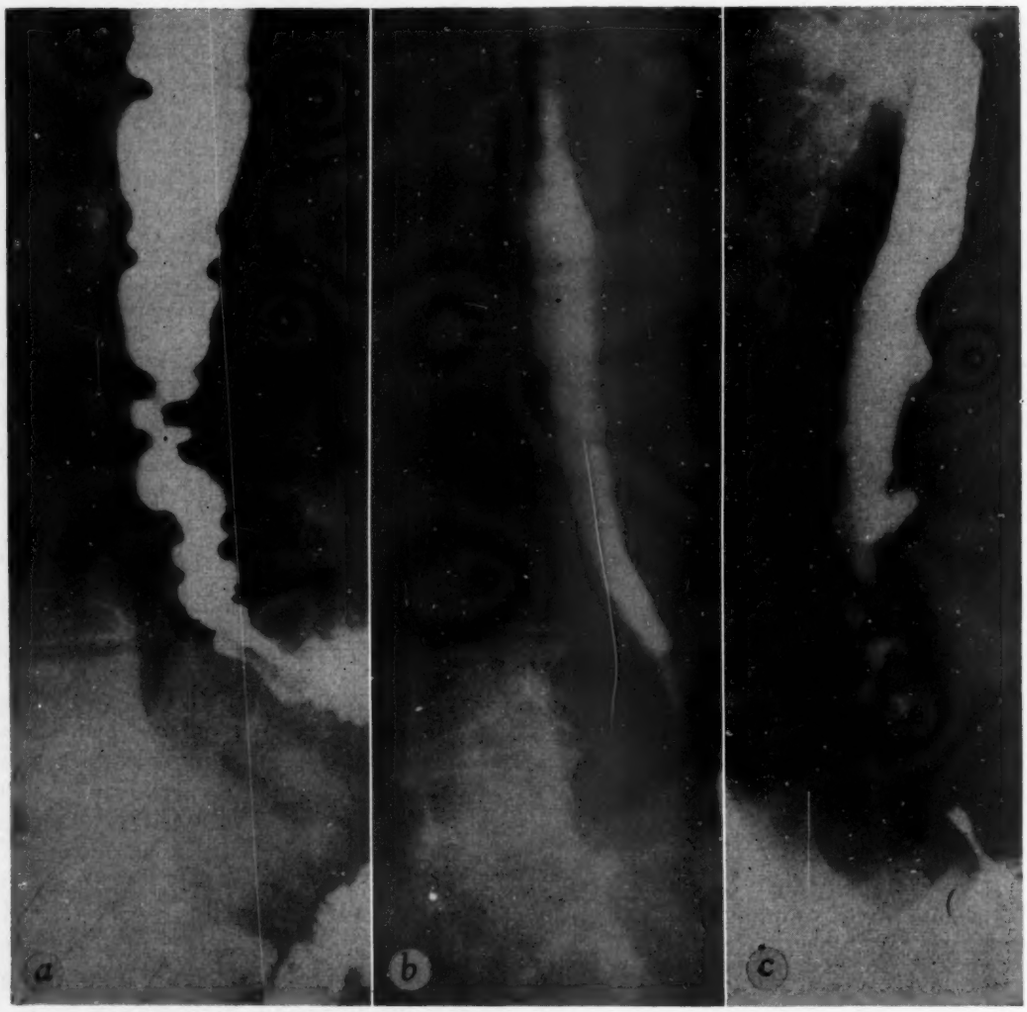

FIG. 3. Diffuse spasm of esophagus. (*a*) Diffuse irregular spasm. (*b*) Diffuse constant narrowing. (*c*) Pseudodiverticulosis. (From: OLSEN, A. M. et al. The treatment of cardiospasm: analysis of a twelve-year experience. *J. Thoracic Surg.*, 22: 164–187, 1951.)

of the associated disease often has a favorable influence on diffuse spasm.

NON-SURGICAL TREATMENT

Satisfactory medical treatment for cardiospasm does not exist. Antispasmodic drugs and sedatives have little effect, and nitrites open the cardia briefly, if at all. Psychiatric therapy is worthless in cardiospasm, although it may be beneficial for patients who have diffuse spasm. Thiamine hydrochloride has not proved helpful in our experience, and orally administered local anesthetic agents apparently do not benefit patients who have achalasia.

Ideally, the treatment of cardiospasm should be directed toward restoration of normal peristalsis. It is unlikely, however, that a means will be found to replace the degenerated ganglionic cells in the esophageal wall. Up to the present time the most effective treatment has consisted of measures that decrease resistance at the cardia. Dilation of the cardia has been employed to great advantage for many years. Passage of bougies (Figs. 4*a* and *b*) usually gives only temporary results. If long-lasting or permanent relief is to be obtained, rather drastic and forceful stretching of the cardia must be done. This usually is accomplished by inflating a properly placed cylindric bag with either air or water. We have used the Plummer hydrostatic bag (Figs. 4*c* and *d*), which is passed over a previously swallowed thread. Permanent relief has been obtained in

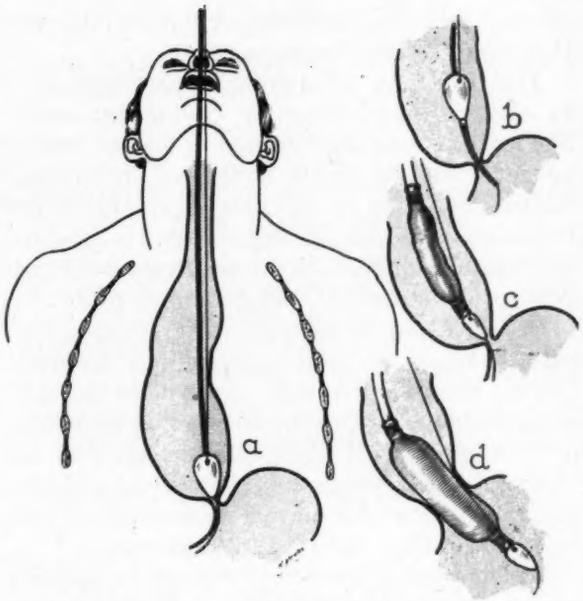

FIG. 4. Method of performing dilation. (*a*) Passage of No. 41 French olive-tipped bougie into stomach. (*b*) A No. 50 or 60 French sound is passed into the stomach guided by flexible wire spiral. (*c*) A hydrostatic dilator is passed to the cardia. (*d*) Distention of the hydrostatic dilator across cardia. (From: OLSEN, A. M. et al. The treatment of cardiospasm: analysis of a twelve-year experience. *J. Thoracic Surg.*, 22: 164–187, 1951.)

60 per cent of our patients with cardiospasm after a single course of such therapy. Of the remaining 40 per cent, about one-half experienced permanent relief after further courses. Thus, 20 per cent either did not respond to this treatment or required periodic dilation.

Hydrostatic dilation of the esophagus is not without some risk. In our series of 601 patients, of whom 555 were treated by means of the hydrostatic dilator, ten had an esophageal "split" [4]. This represents a complication rate of approximately 2 per cent. Fortunately, such complications can be handled satisfactorily by surgical measures and antibiotics in nearly all instances.

SURGICAL TREATMENT

Some form of surgical treatment must be considered for those patients who fail to respond to properly performed hydrostatic dilation. Enthusiasm for a surgical approach to the symptomatic relief of cardiospasm has fluctuated over the years. The many operative procedures that have been and still are being employed attest to the fact that the role of surgery in the management of this disease has not yet been clearly defined.

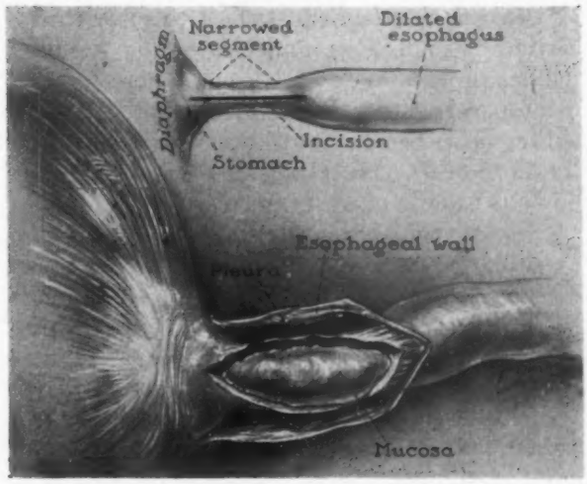

FIG. 5. Esophagomyotomy (modified Heller's procedure).

Among the surgical technics that have been employed are transgastric dilation of the cardia, the Wendel cardioplasty, the Heyrovsky-Gröndahl cardioplasty and various types of esophagogastrectomy.

Cardioplasty, in our experience [26] and that of others [27], has led uniformly to the development of severe ulcerative esophagitis. For this reason, operations of this type should be abandoned. A similar result may follow esophagogastrectomy unless extensive resection of the stomach down to the antrum is done. Such a radical approach to the problem has not appealed to us.

The operative procedure described by Heller [28] and later modified by Zaaijer [29] has given the best results in the surgical management of cardiospasm. This procedure (esophagomyotomy) consists of a linear incision of the esophageal musculature down to the mucosa, carried out in such a fashion as to free completely a portion of the narrowed esophageal segment of its musculature. This operation may be done through the abdomen or transthoracically; we prefer the latter approach. As originally described, the incision was carried well down over the stomach. Since the obstruction is in the esophagus, such a lengthy incision is not necessary and may, in fact, be inadvisable. The incision should be carried onto the stomach only far enough to assure the surgeon that all the esophageal musculature has been divided. (Fig. 5.)

Prior to September, 1956, a total of thirty-seven patients had undergone this modified Heller procedure at the Clinic. There were no postoperative deaths. Ten of these patients either have been operated on too recently for evaluation or have not been available for follow-up examination. Sixteen of the remaining twenty-seven patients have had excellent results; they are asymptomatic, have gained weight and are working. The seven patients who have had good results are also asymptomatic except for occasional dysphagia when nervous or when eating rapidly. The three patients with fair results are improved but have had either recurrence or persistence of some dysphagia. Severe postoperative esophagitis developed in one patient who is classified as having a poor result. Thus, twenty-three of twenty-seven patients (85 per cent) have had good or excellent results.

This clinical impression of improvement is strengthened further by roentgenographic studies showing regression in size of the esophagus toward normal. However, studies of motility continue to show an abnormal pattern characterized by lack of a co-ordinated peristaltic wave.

These early results of esophagomyotomy done on a relatively small number of patients are distinctly encouraging. Such a procedure unquestionably should be done when hydrostatic dilation fails. It cannot yet be stated definitely whether surgical treatment should supplant hydrostatic dilation in the initial management of this disease. The low degree of risk associated with it and the excellent results it affords when properly employed certainly justify its frequent use.

SUMMARY AND CONCLUSIONS

Achalasia of the cardia is a disturbance of esophageal motility characterized by feeble or absent peristalsis and failure of the sphincter to open at the cardia. The cause is not known but when the disease is clinically recognizable it is not a psychosomatic disorder. The diagnosis is suspected when characteristic symptoms are presented; it may be substantiated by roentgenographic studies and endoscopic means, especially by the passage of a blunt olive-tipped bougie over a previously swallowed thread. Physiologic methods that measure esophageal pressures are now available for exact diagnosis of achalasia. Records of motility after injection of methacholine (mecholyl) chloride offer further proof of the diagnosis.

The object of treatment in cardiospasm is to obtain long-lasting or permanent relief. Such results can be obtained in 60 per cent of patients with a single course of hydrostatic dilation and may be ultimately achieved (after two or more courses) in 80 per cent. Hydrostatic dilation is a drastic method of treatment that is not without hazard, yet it is much preferable to half-hearted bouginage with bougies, soft rubber tubes or mercury-weighted balloons. During the course of such inadequate therapy, progressive dilatation of the viscus may take place and valuable time will be lost. The use of a previously swallowed thread, not only for safety but also for proper placement of the hydrostatic bag, deserves re-emphasis.

The success of treatment cannot be entirely correlated with the size of the esophagus but, in general, results of treatment are less satisfactory when the condition is advanced. This statement applies to both endoscopic and surgical methods of treatment. It is obviously unfair to the patient and surgeon to continue with dilation when results are poor and the condition progressive. Thus, if dysphagia is not promptly and completely relieved, surgical treatment should be undertaken. Also, if dysphagia recurs within weeks or months after satisfactory hydrostatic dilation, surgical treatment should be seriously considered.

On the basis of extensive experience we can predict the likelihood of successful treatment with the hydrostatic dilator. We have neither a sufficient number of cases nor a long enough period of observation to provide a statistical analysis of surgical results after cardiomyotomy. However, a properly performed Heller operation probably offers as much to the patient as does hydrostatic dilation, and the risk of complication is probably less.

Thus, there are two good methods of treating cardiospasm. The selection of treatment for the individual patient depends on many factors, including the personal preference of the patient on one hand and the physician on the other. Perhaps the more advanced condition is best managed by esophagomyotomy. It is usually wise to use dilation for early lesions, especially when pain is prominent. The usual patient can be treated either by dilation or surgical intervention. Surgical treatment is employed in many cases if dilation is not satisfactory.

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Foreign Bodies in the Esophagus

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IN a series of 864 cases of foreign body in the esophagus it was interesting to note that approximately half the patients were males and half females (434:430), and furthermore that approximately half the patients were children and half were adults (422:442). The youngest child was a baby only ten days old, who had a safety pin and a button lodged in the cervical esophagus.

In 2,372 cases seen at the author's clinic the most common foreign bodies in numerical order were: (1) bones, 626; (2) coins, 535; (3) food,

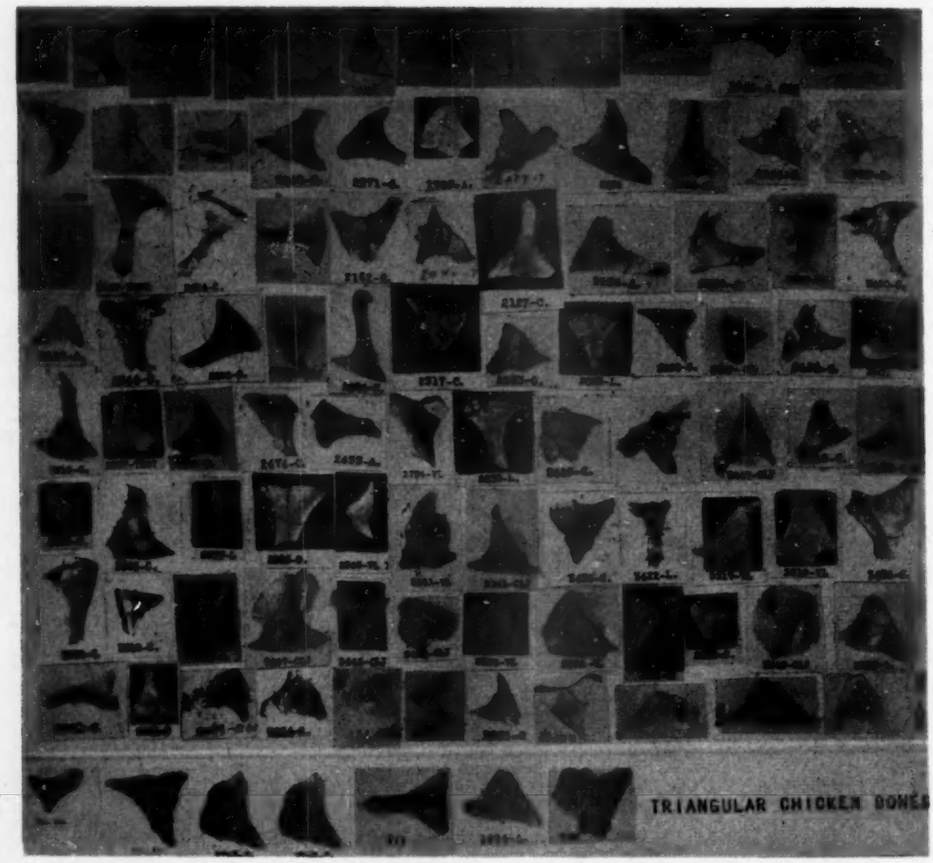

FIG. 1. Bones are the most common foreign bodies found in the esophagus (626 of 2,372 cases). The triangular chicken bones shown in the illustration are especially common.

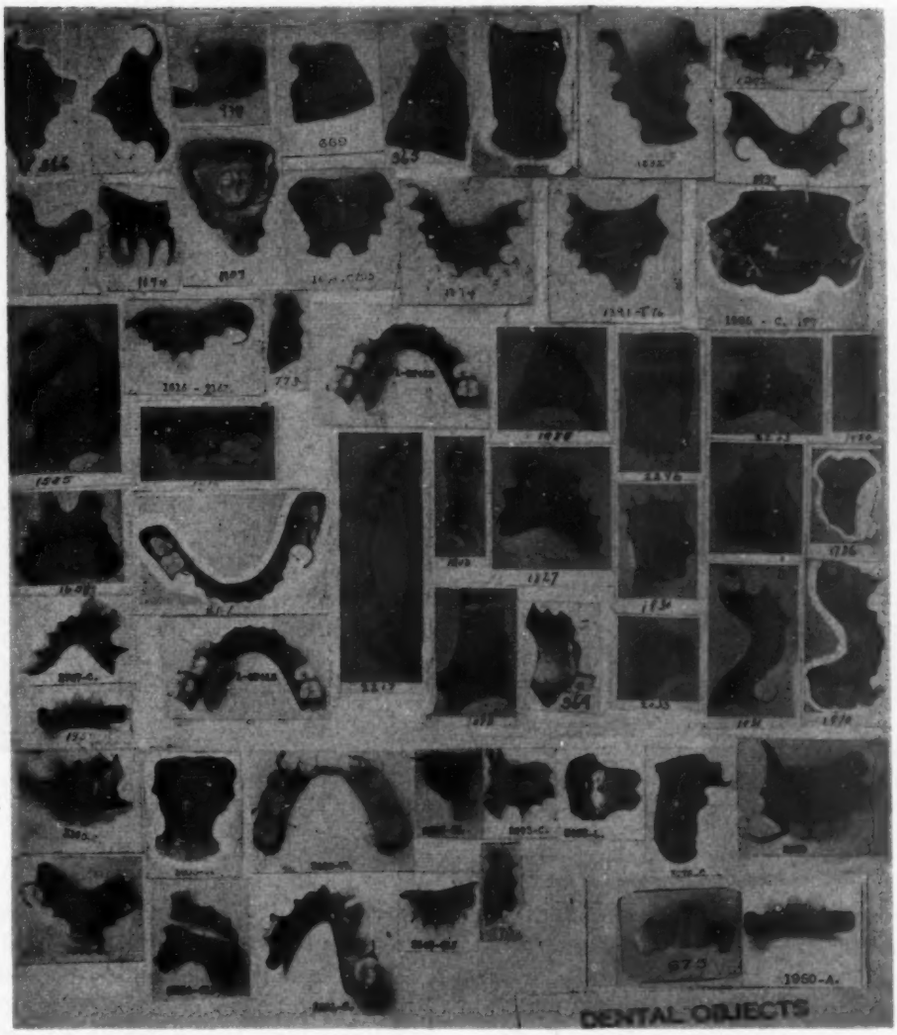

FIG. 2. Dental objects are sixth in frequency in our series of foreign bodies in the esophagus (84 of 2,372 cases). Most common dental objects lodging in the esophagus are artificial dentures.

443; (4) pins, 249; (5) vegetal foreign bodies including nuts, seeds and shells, 97; (6) dental objects, 84; (7) buttons including campaign buttons, 80; and (8) toys, 79. The number of other miscellaneous objects found was 179. (Figs. 1, 2 and 3, Table 1.)

It is not surprising that bones should constitute the largest number of foreign bodies found in the esophagus, since they are normally present in food. The breakdown of coins is interesting: Pennies (237) were most commonly found in the esophagus; the second largest type of coins was nickels (183), and the third, quarters (84). Half dollars, dimes and foreign coins were less commonly found. It is understandable that food should be high on the list, and we found that in 443 cases the foreign body was a fragment of some sort of food. The breakdown of pins is interesting, 231 of the 249 being safety pins; common pins came next (9), bobby pins third (5) and shawl pins fourth (2). Of the vegetal objects, including nuts, seeds and shells, fruit stones were most commonly found (34) and fruit seeds were next in frequency (20). Peanuts and maize numbered 8 each, and among the other objects found were beans, walnuts and almonds. Of the 79 cases of small toys it is interesting to note that 34 were jackstones and 17 were whistles. Other categories included hardware, jewelry, animal shells and minerals.

Etiology. Among etiologic factors in the lodgment of foreign bodies in the esophagus must be mentioned the wearing of artificial

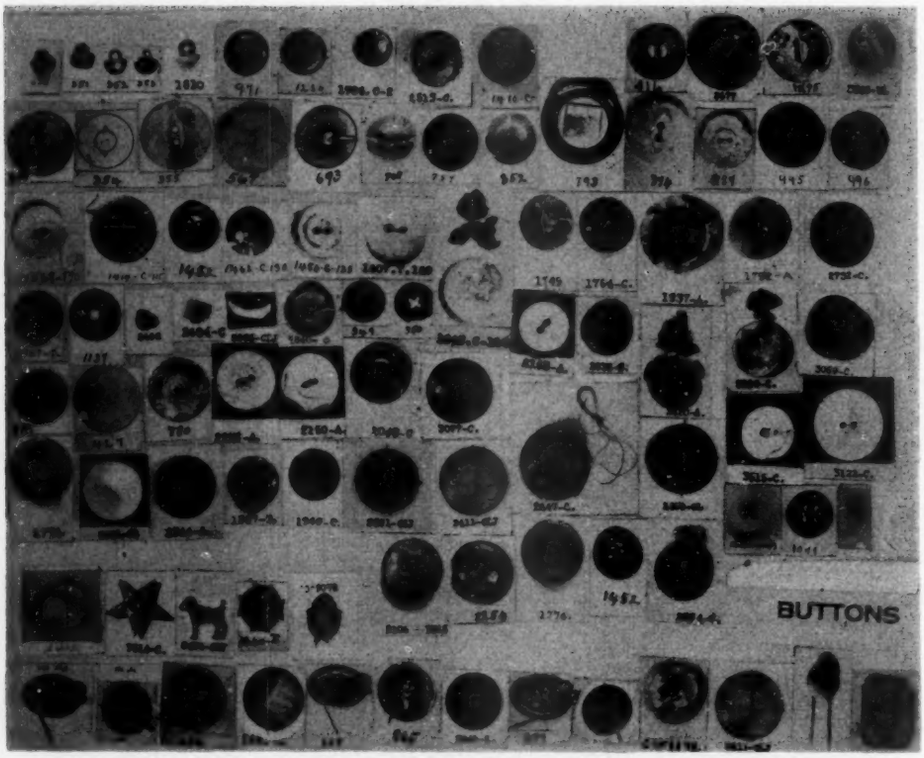

FIG. 3. Buttons rank seventh in incidence among foreign bodies in the esophagus in our experience (80 of 2,372 cases). Campaign buttons are especially dangerous because of the projecting pin.

dentures, because certainly artificial dentures interfere with the detection of a foreign object such as a bone in the mouth. Secondly, carelessness in the preparation of food must be mentioned as an important etiologic factor. Hasty, careless eating may be a factor. An obvious causative factor is the placing of inedible objects such as coins and buttons in the mouth.

The existence of stenosis or a stenosing lesion such as a tumor will, of course, predispose to lodgment of an esophageal foreign body. This is one of the reasons that an "after-ray" should always be taken following the removal of a foreign body; and if there is any suspicion of esophageal abnormality, diagnostic esophagoscopy should be done before the patient is discharged.

Symptomatology. The symptoms of foreign body in the esophagus are choking and gagging, sensation of lodgment of a foreign object, and pain in the case of sharp-pointed foreign bodies. Complete obstruction with inability to swallow may be produced by lodgment of a large foreign body. Occasionally a large foreign body in the esophagus can give sufficient compression of the airway to cause shortness of breath, and not infrequently the spill-over of secretions into the laryngotracheal airway will cause cough.

Diagnosis. Diagnosis of a foreign body in the esophagus is made on a basis of the history of swallowing a foreign object, together with confirmative x-ray examination with or without the use of an opaque mixture, and by the presence of the aforementioned symptoms, though the symptoms may be slight or absent. (Fig. 4.)

TABLE 1

Bones	626
Coins	535
Food	443
Pins	249
Vegetal objects, e.g., nuts, seeds and shells	97
Dental objects	84
Buttons, including campaign buttons	80
Toys	79
Other objects	179
Total	2,372

Treatment. The only treatment for foreign

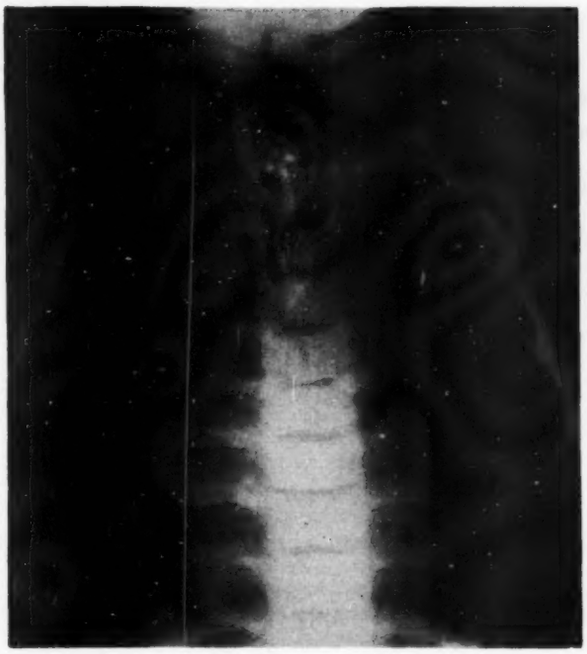

FIG. 4. Some buttons are opaque to the x-ray; many are not. In the case of non-opaque buttons the use of an opaque mixture is necessary for visualization, as in the case of the casein button illustrated above.

FIG. 5. Manner of grasping the esophagoscope.

body in the esophagus is, obviously, removal of the foreign body. This can be accomplished by esophagoscopy in practically all cases. The only exceptions are cases of very large, tightly impacted foreign bodies. In rare instances these may require external operation (esophagotomy).

The technic of esophagoscopy is simple, but certainly the safe introduction of the esophagoscope requires a certain amount of training and experience. General anesthesia is rarely necessary but the patient should receive appropriate premedication and then should be placed in the proper position with his head held correctly. For introduction of the esophagoscope the patient's head should be held high and his shoulders maintained flat on the table. The esophagoscope is grasped in the manner illustrated in Figure 5 and introduced first into the right pyriform sinus. Lifting the larynx forward with the thumb of the left hand and directing the tip of the esophagoscope toward the suprasternal notch, it is necessary to wait for the cricopharyngeus muscle to relax and the esophagus to open. Relaxation of this muscle and opening of the esophagus can be facilitated by the introduction of a small flexible bougie. Care should be taken not to override the foreign body, which is most commonly located just below the "cricopharyngeal pinchcock." When the foreign body is visualized, the location of best "forceps spaces" should be noted and the appropriate forceps introduced. When a secure grasp is obtained, the esophagoscope is advanced to contact with the foreign body and forceps, foreign body and esophagoscope gently withdrawn together. In the case of an irregular, sharp-pointed object the right angle rotation forceps is used; in this instance it is best not to hold the foreign body close to the end of the esophagoscope, so that a certain amount of free rotation may take place and the sharp points be thus prevented from inflicting trauma.

In every case it is desirable to obtain a duplicate of the foreign object for study and practice. Study of the duplicate enables us to select more intelligently the best forceps to use and also affords an opportunity to study the various possible presentations. Obviously, in the case of bones or fragments of food it will probably not be possible to obtain a duplicate; however, practice may be obtained by working with objects of similar character and varying size and shape.

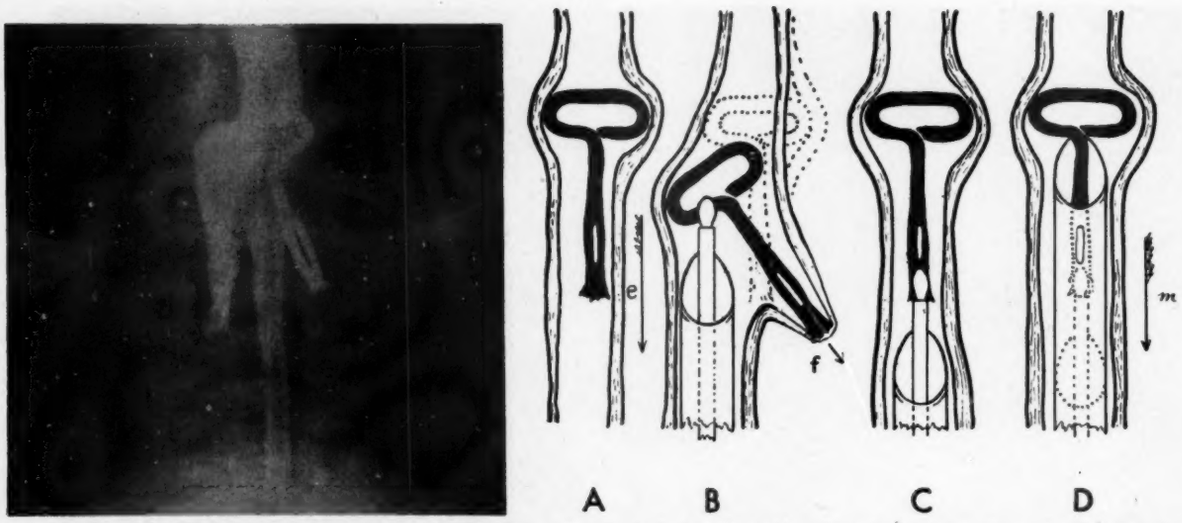

FIG. 6. Key can opener lodged in the cervical esophagus. Perforation is easily caused, as shown in x-ray film at the left, if traction is made on the lower end or on the shaft instead of at the presenting extremity. In dealing with a foreign body, attention must always be paid to the particular mechanical problem presented. The danger of perforation and the way to avoid it are clearly shown in the schema to the right. Both x-ray and schema are intentionally placed upside down in order to show the same relations as those presented in the patient on the operating table.

Complications. In the early days of esophagoscopy, complications from the mere introduction of the esophagoscope were frequent. Perforation of the hypopharynx or the esophagus occurred in a relatively high percentage of cases and the consequences were grave. At the present time, with improved technic of esophagoscopy, perforation is much less common; and with antibiotic therapy available, even when perforation does occur, the outlook is less grave. Perforation is manifested by the presence of subcutaneous emphysema or by mediastinal emphysema as shown in the x-ray. If perforation has taken place, either by the instrument itself or by the foreign body, antibiotic therapy should be instituted at once; and if response is not satisfactory, cervical mediastinotomy is indicated. (Fig. 6.) Generally, if the foreign body has not been removed, its removal should be postponed until the subsidence of acute reaction.

Propbylaxis. The lodgment of esophageal foreign bodies can be prevented by the exercise of care in the preparation of food, by taking special care to detect the presence of foreign objects such as bones in food for persons wearing artificial dentures, and by the avoidance of putting inedible objects such as coins and buttons into the mouth.

Prognosis. The prognosis of foreign body in the esophagus should be excellent. As indicated heretofore, virtually any foreign body lodged in the esophagus can be removed esophagoscopically, and even in the rare cases in which esophagotomy may be required the outlook is good with modern surgical technic.

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Bleeding Esophageal Varices

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MASSIVE bleeding from esophageal varices is one of the most serious problems presented to the surgeon. Esophageal varices almost always indicate portal hypertension.

The esophagus becomes involved in the problem of portal hypertension because of the anatomic relationship of the esophageal veins and the extrahepatic portal circulation. (Fig. 1.) The portal vein is formed by the junction of its two main tributaries, the splenic and mesenteric veins. These veins are without valves. Obstruction to the portavenous flow, whether intrahepatic or extrahepatic, may actually produce a reversal of the flow in the tributaries of this valveless venous system. The body attempts to compensate for this increased flow by a series of collateral vessels, diverting the blood from the portal system into the general venous system, actually forming a natural portavenous shunt. The collateral circulation which concerns us is that involving the esophageal veins. (Fig. 2.) This natural shunt links the vasa brevia of the left gastric and coronary veins by way of the esophageal veins to the azygos and hemiazygos veins, thence to the superior vena cava and general venous circulation. When such natural compensatory portacaval shunts are not sufficient to lower the portal pressure and when it is remembered that the dilated esophageal varices are exposed to direct trauma of food and peptic regurgitation of acid gastric secretions, it is understandable why they erode or lacerate and massive hemorrhage ensues. It is the vulnerability of this natural compensatory collateral circulation that brings portal hypertension to the attention of the surgeon.

The causes of esophageal varices are the same as the causes of portal hypertension. They are the result of prehepatic, intrahepatic or extrahepatic block. (Table 1.) In about 80 per cent

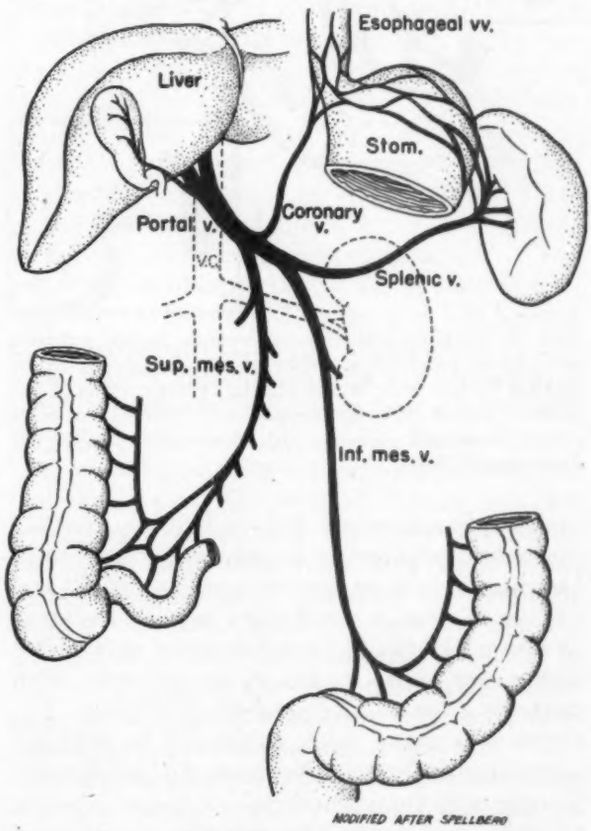

FIG. 1. Extrahepatic portal circulation.

of the cases, esophageal varices are associated with Laennec's cirrhosis and the majority of the remaining cases fall into the so-called Banti's extrahepatic group. In discussing the diagnosis and treatment of esophageal bleeding

from ruptured varices, the underlying pathology of the portal hypertension must constantly be kept in mind.

CLINICAL EVALUATION

The clinical manifestation of massive upper gastrointestinal hemorrhage is quite evident—

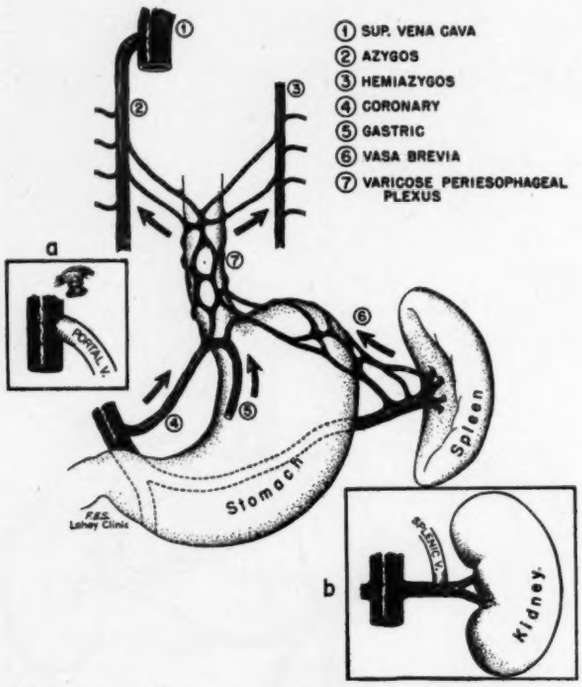

FIG. 2. Note reverse flow of venous blood, nature's attempt to establish a collateral portal systemic shunt linking up the vasa brevia and left gastric veins of the portal system with the azygos and hemiazygos veins of the systemic system. (*a*) Portacaval shunt. (*b*) Splenorenal shunt.

massive hematemesis followed by shock. The diagnostic problem is to determine the site of bleeding. Is it secondary to ruptured esophageal varices or does it result from peptic ulceration or tumor? It should be remembered that peptic ulcers are more common in patients with cirrhosis than in the general population. Patients who have been subjected to subtotal gastrectomy for massive hemorrhage thought to originate from duodenal ulcer have subsequently died from hemorrhaging esophageal varices. Likewise, patients have died from a bleeding duodenal ulcer while all attention was directed toward enlarged esophageal varices. A history of upper gastrointestinal distress or ulcer is helpful. A history of alcoholism, dietary deficiency and infectious hepatitis may suggest esophageal varices. In esophageal bleeding fresh blood and clots are seen, whereas in cases of bleeding from the stomach, because of certain changes in the blood, it appears to be typical coffee-ground vomitus.

Clinical examination may reveal evidence of liver damage, jaundice, ascites, spider nevi or caput medusae, thus suggesting liver disease, portal hypertension and bleeding esophageal varices. An enlarged spleen immediately directs attention to the possibilities of an extrahepatic block, formerly called Banti's disease. If the diagnosis is not certain after clinical examination, a barium roentgenologic study should be made. I do not believe that small amounts of thin barium by mouth will activate esophageal or peptic bleeding and to ascertain the correct diagnosis and thereby institute the correct therapy it is well worth the slight risk, if any, that is involved. The roentgenogram does not always give the true status of esophageal varices; and if varices are suspected, we follow roentgenologic examination with esophagoscopy. The size of varices as seen on the roentgenogram is no indication that they are bleeding. Another diagnostic tool used to determine the site of upper gastrointestinal hemorrhage is the Sengstaken tube which helps control

TABLE 1
CAUSES OF ESOPHAGEAL VARICES*

I. With portal hypertension
  A. Intrahepatic
    1. Laennec's cirrhosis
    2. Postnecrotic cirrhosis
    3. Toxic cirrhosis
    4. Syphilitic cirrhosis
    5. Schistosomiasis
    6. Congenital intrahepatic portal stenosis
  B. Extrahepatic
    1. Thrombosis of portal or splenic veins (acute infection, adhesions, lymph nodes, tumors, pancreatitis)
    2. Compression of portal vein
    3. Stenosis of portal vein
    4. Arteriovenous aneurysm
  C. Suprahepatic
    1. Right heart failure with cardiac cirrhosis
    2. Thrombosis of hepatic vein
  D. Obstruction of azygos venous system
II. Without portal hypertension

* Modification of classification by RACK, F. J., MINCKS, J. R. and SIMEONE, R. Observations on etiology of esophageal varices. *Arch. Surg.*, 65: 422–429, 1952.

bleeding by the pressure of the balloon against the esophageal varices. Once the esophageal bleeding is controlled, the aspirations from the stomach should be free of blood.

LABORATORY INVESTIGATION

Hematology. The status of the blood as determined by the leukocyte count may reveal pancytopenia. This, coupled with a palpable spleen, indicates what we now call hypersplenism, and a white blood cell count below 2,000 and platelets numbering less than 150,000 would be expected. The obvious anemia may be from the bleeding rather than from the hyperactivity of the spleen.

Liver Function Tests. Evaluation of liver function is important not only in helping with the diagnosis but also in choosing candidates for surgery. Several liver function tests are done so that we know as much as possible about the capacity of the liver. We attach the most significance to the following: (1) The blood protein level (mainly the serum albumin) indicates the capacity of the liver to manufacture proteins. Collective series have shown that the operative mortality in shunt procedures is greatly increased when the serum albumin is under 3 gm. (2) The bromsulphalein® test measures the capacity of the liver to eliminate abnormal substances. This should be under 15 per cent to make the operation safe. (3) The prothrombin time, or the capacity of the liver to manufacture prothrombin, should be 60 per cent or greater. We are also interested in the response of the liver to vitamin K. If the prothrombin is 60 per cent and after the administration of vitamin K it increases to 100 per cent, we are encouraged regarding the function of such a liver. (4) Cephalin flocculation and thymol turbidity are tests for gamma globulins. They may be unreliable in the presence of a hyperactive spleen. The readings should be no greater than 2 plus before operation. (5) The serum bilirubin and the alkaline phosphatase values are determined to rule out the presence of biliary obstruction.

Liver biopsy is frequently performed as an aid in diagnosis.

Paracentesis helps in preparing the patient for surgery and also gives the doctor an opportunity to examine the fluid to make sure that the symptoms are not due to some other cause such as generalized carcinomatosis.

An intravenous pyelogram is always obtained so that if the surgeon decides to perform splenorenal anastomosis at the time of operation, he knows that both kidneys are functioning properly.

PREOPERATIVE PREPARATION

The preoperative preparation of the patient with bleeding esophageal varices and poor liver function involves: (1) control of the acute phase of hemorrhage, (2) replacement of blood volume and (3) measures to improve the liver function. In the case of acute, uncontrollable bleeding the patient is given large quantities of whole blood. If the bleeding does not subside spontaneously, as it usually does in young persons and in those who have experienced only one or two such episodes, it may be necessary to attack the bleeding source itself. The acute bleeding may be stopped either by the use of the Sengstaken tube or by direct transpleural ligation of the vessels involved. In most instances the patients can be tided over by the use of the tube and this, I believe, is the procedure of choice. Once the hemorrhage has been controlled and the blood volume returned to normal, the remainder of the preoperative therapy consists of improving the functional state of the liver. If the serum albumin level is low, serum albumin is given intravenously. The patients are maintained on a soft diet to prevent further bleeding. The diet is rich in protein and carbohydrates and is supplemented with liver extract and vitamins. If liver function is poor it is always difficult to make a decision regarding definitive surgery. We know that the risk is much greater when liver function is poor and that if prolonged medical therapy is given it will improve, but we do not know whether an exsanguinating hemorrhage may occur while the patient is being prepared for definitive surgery. Careful judgment is necessary to make the correct decision and frequently it cannot be made. In general, the more time taken before surgery to prepare a patient with poor liver function, the better the results will be.

Recent work by McDermott and associates [1], and emphasized by Welch and his coworkers [2], indicates that hepatic coma may partially be the effect of ammonia intoxication. It is thought that a high blood level of ammonia is secondary to absorption of blood from the gastrointestinal tract. Welch suggested purgatives and colonic enemas to rid the gastrointestinal tract of massive amounts of blood.

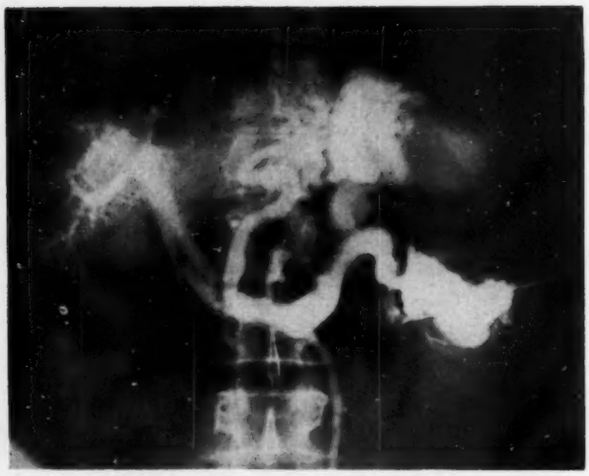

FIG. 3. Typical splenoportogram with biliary cirrhosis. Note large splenic and portal veins and also coronary vein leading to esophageal varices.

Furthermore, if the blood ammonia level is elevated, the sodium salt of l-glutamic acid may be given. This salt unites with the ammonia to form glutamine which is metabolized without the formation of ammonia. In this way high ammonia levels may be lowered and the effects of ammonia intoxication diminished.

SELECTION OF PROCEDURE

What procedures do we recommend once the patient is ready for definitive surgery? We have a choice of splenorenal anastomosis, portacaval anastomosis or esophagogastrectomy. In cases of extrahepatic block, in which the portal vein cannot be used satisfactorily, splenorenal anastomosis must be done. In the vast majority of cases of intrahepatic blocks, however, portacaval anastomosis rather than splenorenal anastomosis is indicated. With this shunt a larger anastomosis can be made. It is fair to assume that the larger anastomosis will divert more blood from the hypertensive portal system and therefore produce a lower portal pressure. Furthermore, with the larger shunt there is a better chance that it will remain patent. As mentioned earlier in the discussion, many patients with portal hypertension and cirrhosis have associated splenomegaly and pancytopenic purpura or hypersplenism. In such cases it may be better to remove the spleen and improve the blood picture of the hypersplenism. Splenectomy is indicated in these patients and inasmuch as the operation is performed on the left side of the thorax and abdomen, splenorenal anastomosis should be done.

It should be pointed out that before the decision is made to do splenorenal or portacaval anastomosis the status of the splenic and portal veins should be noted. With the advent of splenoportography [3, 4] this is possible. (Fig. 3.) A radiopaque material may be injected percutaneously directly into the spleen and a roentgenogram taken. We will then know preoperatively whether or not a splenic or portal vein is available which is adequate for anastomosis, and the procedure can be planned accordingly. The routine at this time is to anesthetize and intubate the patient to control respiration, do the splenoportogram and then proceed immediately with the operation. We have been hesitant to carry out splenoportography without anesthesia and without immediately opening up the abdominal cavity because on some occasions following splenoportography we have found several hundred cubic centimeters of blood in the abdominal cavity at the time of operation. The splenogram is obtained by the percutaneous injection of 50 cc. of 70 per cent urokon® or diodrast® through the eighth or ninth interspace directly into the spleen. The splenogram usually gives sufficient information relative to the splenic and portal veins. If the splenogram is unsatisfactory it is a simple matter to obtain a portal portogram by injecting diodrast directly into the portal system. Once the abdomen is open, a small cannula is inserted into the branches of the superior mesenteric vein in all cases to determine the portal pressure. If the splenogram is not satisfactory after taking the portal pressure, another 50 cc. of 70 per cent urokon is injected directly into the cannula, a roentgenogram is taken, and the status of the portal vein determined. We can then decide whether to do a portacaval or splenorenal shunt.

The third procedure, esophagogastrectomy, is reserved for those patients with esophageal bleeding whose portal or splenic vein is not satisfactory for anastomosis.

In recent years we have been confronted with the patient who has esophageal bleeding several years after splenectomy. Usually he is young, with an extrahepatic block, and has previously undergone splenectomy without a splenorenal anastomosis for esophageal bleeding. A portogram may reveal that the portal vein is absent or abnormal. (Fig. 4.) Such patients,

FIG. 4. Postsplenectomy bleeding esophageal varices. Portogram shows absence of splenic portal veins. Esophagogastrectomy was performed.

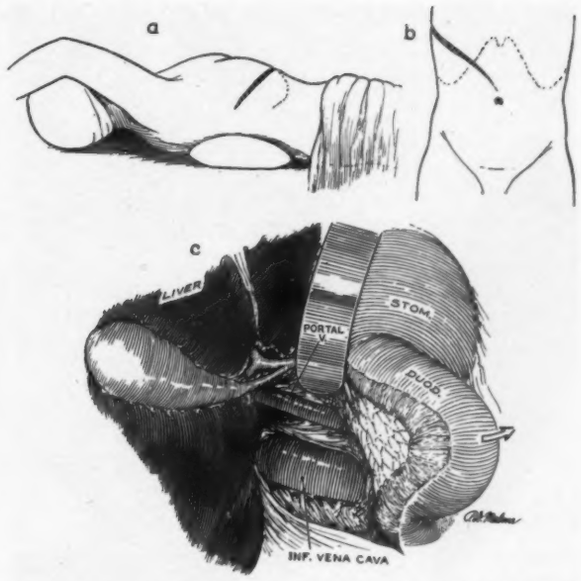

FIG. 5. (*a*) and (*b*) Long, curved, thoracoabdominal incision with patient in semi-lateral position. In (*c*) note postparietal peritoneum is incised, vena cava and portal vein are exposed and duodenum is mobilized. (From: SEDGWICK, C. E. and PARRISH, C. M. Portal hypertension. *S. Clin. North America*, p. 673, 1955.)

after emergency methods have been used to control acute hemorrhage, will benefit from esophagogastrectomy.

PROCEDURE

Portacaval Shunt. We believe the best exposure for the portacaval shunt is obtained with the patient in a semi-lateral position. A long oblique thoracoabdominal incision is made, extending from just above the umbilicus through the eighth interspace well posterior to the lumbar muscles. (Fig. 5.) The diaphragm is divided, the lung is packed above and the liver, which is usually large, is displaced upward partially into the chest cavity. Manometric portal pressure readings are obtained and portal portography is performed if the splenoportogram was unsatisfactory. If a portal vein is seen, surgery is continued. The stomach and bowel are packed to the left and cephalad. The hepatic flexure is freed and retracted downward; the posterior parietal peritoneum is excised laterally and dissection is continued medially, thereby exposing the vena cava at about the level of the renal vein. About two-thirds of the anterior circumference of the vena cava is mobilized. It is not necessary to mobilize the vena cava completely posteriorly. In this way the lateral posterior lumbar veins are not disturbed, and troublesome bleeding is avoided.

Attention is next directed to the portal vein which is identified and mobilized on the posterior border of the porta hepatis. It should be noted that frequently there is a mass of lymphoid tissue overlying the porta hepatis which bleeds easily and profusely and makes identification of the portal vein difficult. In some cases it may be easy to excise this lymphoid tissue immediately and thereby obtain better exposure of the portal vein more quickly. As much portal vein as possible should be mobilized and this can be obtained by mobilization of the duodenal sweep and head of the pancreas. Occasionally a large caudate lobe of cirrhotic liver may separate the portal vein from the vena cava by as much as 7 or 8 cm. The portal vein is then ligated and transfixed as high as possible in the hilus of the liver. There are many types of vascular clamps for occlusion or partial occlusion which may be used on the portal vein and vena cava. (Fig. 6.) For the portal vein, I prefer the clamp devised by Linton which allows occlusion of the vessel at the highest possible level. It is easily applied and the handle is angulated so as not to obstruct

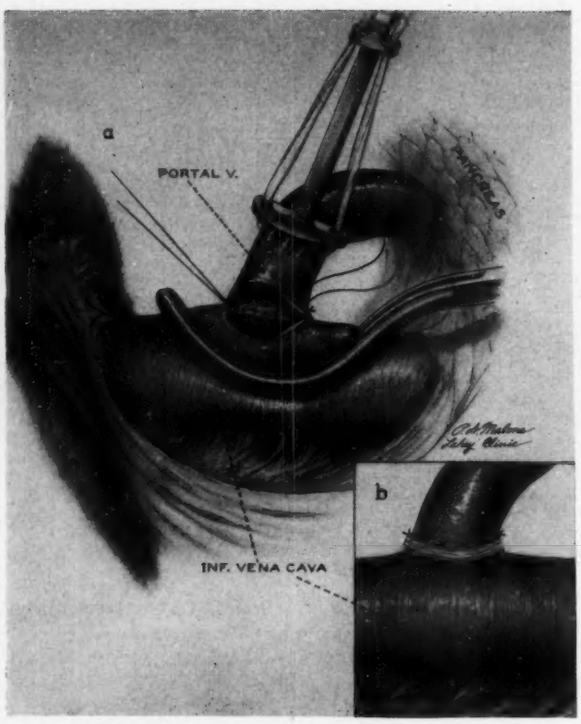

FIG. 6. Portacaval anastomosis. (*a*) Note high ligation of portal vein and Linton clamp on vein. Posterior aspect of vena cava is not completely immobilized. Clamp on vena cava produces partial occlusion. (*b*) Completed anastomosis. (From: SEDGWICK, C. E. and PARRISH, C. M. Portal hypertension. *S. Clin. North America*, p. 674, 1955.)

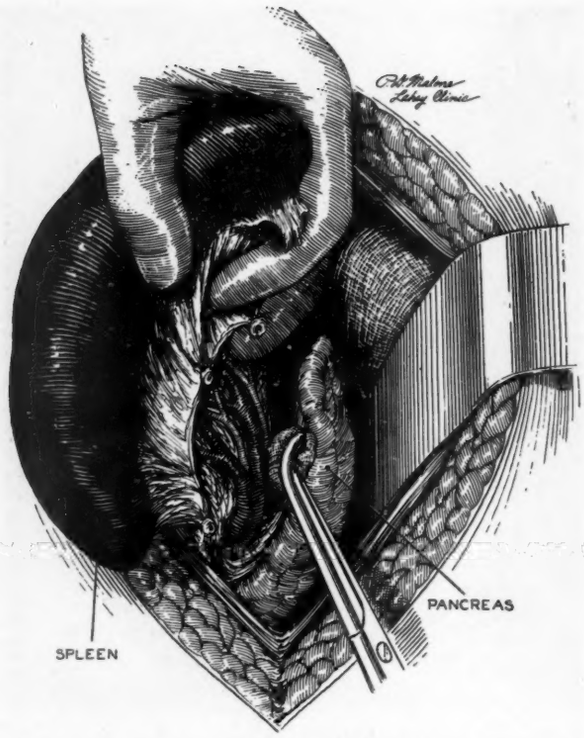

FIG. 7. Spleen is completely mobilized and retracted out of the wound, and splenic vein is isolated from behind. (From: SEDGWICK, C. E. and PARRISH, C. M. Portal hypertension. *S. Clin. North America*, p. 676, 1955.)

the field during the anastomosis. Several clamps are available which are satisfactory for partial occlusion of the vena cava. Anastomosis is made with interrupted everted sutures of No. 5-0 Deknatel silk.

At the conclusion of the anastomosis a good pulsation is felt as the portal blood rushes into the vena cava. A drain is placed in Harrison's pouch, a catheter in the chest, and the abdomen is closed.

Splenorenal Shunt. If we decide to do a splenorenal instead of a portacaval shunt, the same type of thoracoabdominal incision is made except that it is on the left side. When the abdomen is open and the portal pressure is recorded, the lesser sac is entered, the stomach is retracted upward, completely freed along the greater curvature, and the short gastric arteries are divided. The transverse colon and the splenic flexure are separated from any attachments to the splenic pedicle and retracted downward. The splenic artery is then isolated along the superior border of the pancreas and ligated. The splenic vein is approached from behind and separated from the pancreas by meticulous division and ligation of the many small tributaries of the splenic vein coming from the pancreas. (Fig. 7.) All precautions are taken to obtain as long a segment of splenic vein as possible. When the necessary segment of splenic vein is isolated, the remaining vascular connections to the hilus of the spleen are divided and the spleen is removed. It should be emphasized that the spleen is not removed at the beginning of the procedure as it acts as a retractor and helps with the mobilization of the splenic vein. Attention is directed to the kidney. The posterior parietal peritoneum is divided just medial to the hilus of the kidney and the renal vein is isolated. The kidney is not mobilized. Usually the ovarian and adrenal veins must be ligated. The venous outflow is occluded temporarily by heavy silk ligatures. (Fig. 8.) The anastomosis is made the same way as with the portacaval shunt; the end of the splenic vein is anastomosed to the side of the renal vein using interrupted everted mattress sutures.

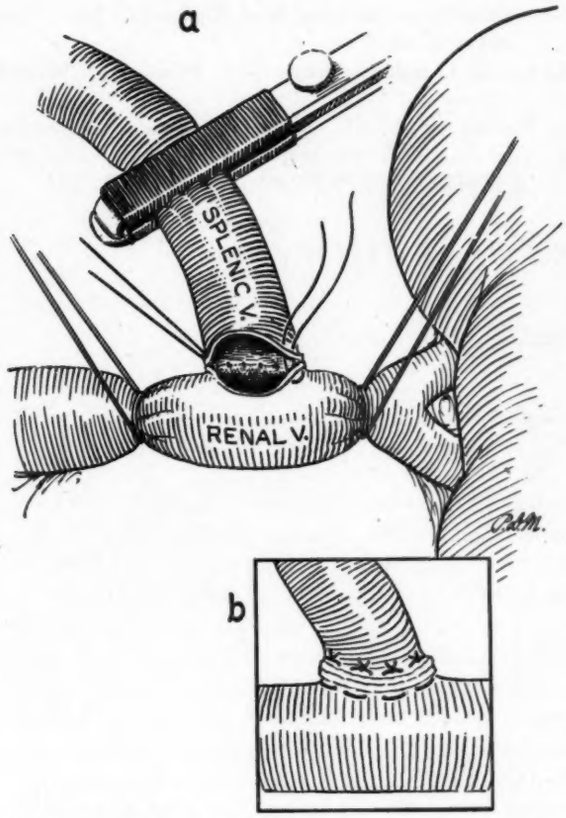

FIG. 8. (*a*) Renal vein is controlled with heavy silk. (*b*) Interrupted everting sutures are utilized for the anastomosis. (From: SEDGWICK, C. E. and PARRISH, C. M. Portal hypertension. *S. Clin. North America*, p. 677, 1955.)

Esophagogastrectomy. Esophagogastrectomy consists of resection of the lower third of the esophagus and the upper third of the stomach. Theoretically, it might be assumed that it is better to perform esophagojejunostomy to prevent gastric regurgitation but we have not found this necessary. We excise the upper third of the stomach, make a tube from the remaining portion of stomach and anastomose this to the esophagus. (Figs. 9 and 10.)

POSTOPERATIVE CARE

In the postoperative care it is important to prevent distention and reaccumulation of fluid. The usual precautions following thoracotomy are adhered to. Anticoagulants are not used. Terramycin,® 500 mg. a day, is given the first five or six postoperative days. The patients are encouraged to take nourishment by mouth as soon as possible. Abdominal distention is watched for and is dealt with by means of enemas and administration of prostigmin.®

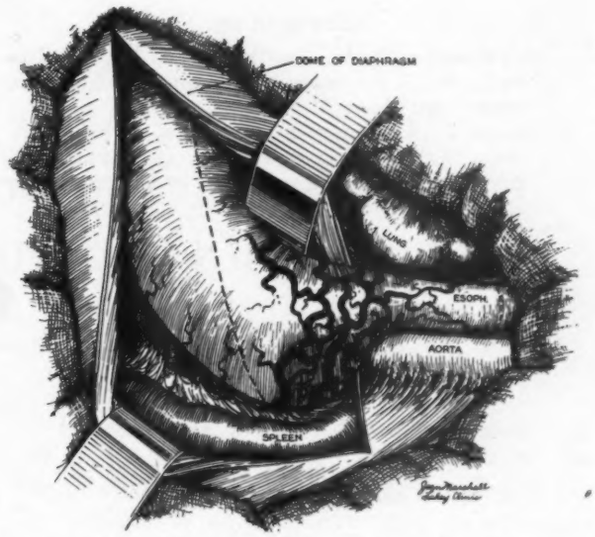

FIG. 9. Esophagogastrectomy. Dotted line indicates area to be resected. (From: ADAMS, H. D. Treatment of portal hypertension and associated acute hemorrhage. *S. Clin. North America*, p. 806, 1951.)

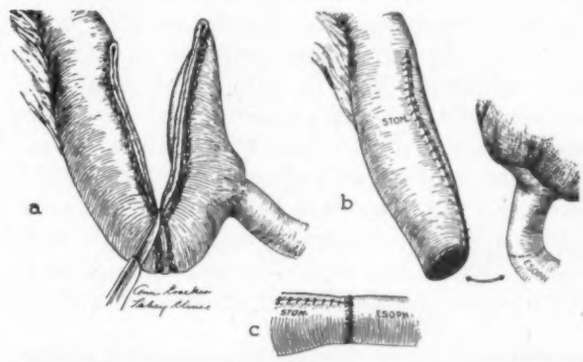

FIG. 10. (*a*) Stomach and lower esophagus resected. (*b*) A tube is fashioned from the antrum of the stomach. (*c*) Esophagogastric anastomosis.

SUMMARY

Esophageal varices are the result of portal hypertension. Bleeding from varices occurs as the result of nature's attempt to shunt the high portal pressure of the portal bed into the systemic veins. Acute bleeding may be controlled for a short period of time either by the Sengstaken tube or by direct transthoracic ligation of the involved veins. Definitive surgery consists of reducing portal pressure by diverting blood from the portal to the general venous system by a splenorenal or portacaval shunt. If a shunt procedure is impossible, esophagogastrectomy must be performed. Therapy directed toward improving liver function is of paramount importance.

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Acquired Non-malignant Esophagorespiratory Fistula

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THE acquired non-malignant esophagorespiratory fistula is rare, and its infrequent occurrence allows little experience for the physician desiring to manage it. Coleman and Bunch [4] were able to collect from the literature over a period of thirty-two years, 1916 to 1949, only seventy-five cases of acquired non-malignant fistula. A subsequent review of only the English literature to July, 1956, discloses eighteen additional cases. Experience with six fistulas of this variety and a study of the reported cases prompts reconsideration of the incidence, clinical features and management of this type of lesion.

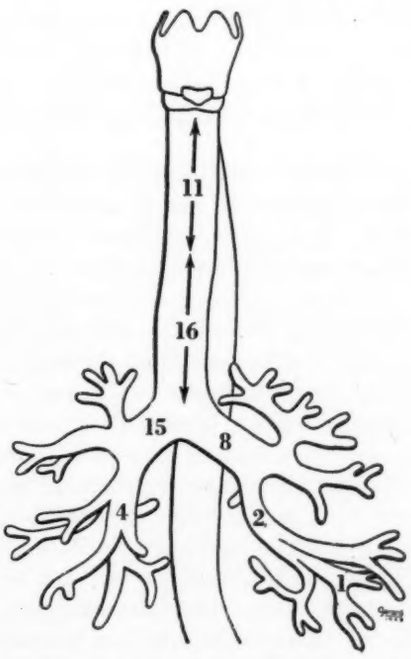

FIG. 1. Anatomic location of esophagotracheobronchial fistula in fifty-seven reported cases.

The anatomic relationship of the esophagus to the respiratory tract is conducive to the development of fistulas, as the esophagus lies in immediate contact with the membranous portion of the trachea and the left stem bronchus. An analysis of the reported acquired non-malignant esophagorespiratory fistulas reveals fifty-seven case reports in which the location of the fistula is given. (Fig. 1.) In this group the esophagus communicated with the right stem bronchus more often than the left. This may be peculiar to only the benign type of fistula, for previous reports of the acquired variety, malignant and non-malignant, show that the fistula develops more frequently between the esophagus and left stem bronchus. The deep cervical, paratracheal and tracheobronchial groups of lymph nodes lie in immediate contact with the esophagus, trachea and

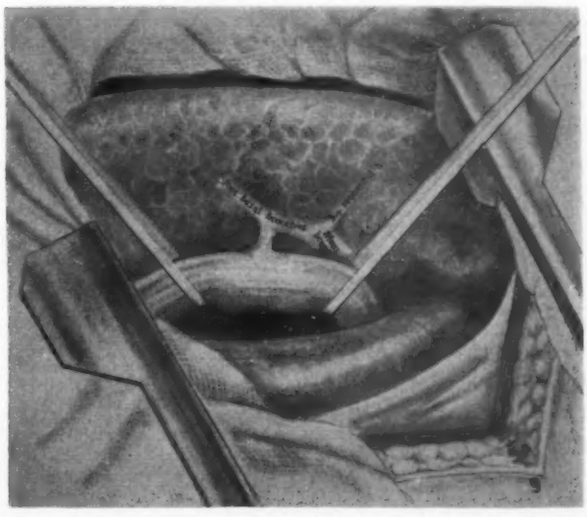

FIG. 2. Fistulous communication between the esophagus and posterior basal segmental bronchus of lower lobe of left lung. (From: COLEMAN, F. P. and BUNCH, G. H., JR. Acquired non-malignant esophagotracheobronchial fistula. *J. Thoracic Surg.*, 19: 542, 1950.)

major bronchi, and likely play an important role in the pathogenesis of fistulas of infectious origin. (Fig. 2.)

ETIOLOGY

Malignant lesions account for 50 to 60 per cent of all acquired communications between the esophagus and respiratory tree [9,11]. Acquired non-malignant esophagorespiratory fistulas may best be classified on the basis of etiology. (Table I.) In an analysis of ninety-three reported cases, trauma was found to be their most common cause. Of the thirty-one traumatic fistulas, nine were caused by blunt force or non-penetrating injury to the thorax. (Table II.)

TABLE I
CLASSIFICATION OF ESOPHAGOTRACHEOBRONCHIAL FISTULAS

I. Congenital
II. Acquired
  A. Malignant
  B. Non-malignant
    1. Traumatic
      a. Foreign body
      b. Instrumentation
      c. Wounds of thorax
      d. Chemical (ingestion of caustics)
    2. Esophageal diverticulum
    3. Infection
      a. Tuberculosis
      b. Syphilis (gumma or aneurysm)
      c. Fungus
      d. Non-specific

There is a correlation between the traumatic agent and the site of communication of the fistula with the respiratory tract. The fistulas caused by compression injury communicated with the trachea in seven of the nine patients, while those caused by foreign bodies and instrumentation of esophageal strictures communicated with either the right or left stem bronchus in eight of nine patients. With the exception of the case due to blast injury cited by Volk et al. [13], the nine traumatic fistulas due to blunt force developed between the esophagus and membranous portion of the terminal trachea or proximal membranous portion of the stem bronchus.

It is frequently difficult to arrive at the specific type of infection which is responsible for a fistula between the esophagus and respiratory tract. Formation of a fistula with epithelization of its tract is a latent sequela of mediastinal infection, and even with excision of the tract, there is often insufficient evidence to identify the etiology of the fistula. In a study of ninety-three reported cases of non-malignant fistulas, twenty-eight were known to be caused by a specific type of infection. (Table III.) Although in nineteen additional cases the etiology of the fistula could not be determined by history, physical findings and frequently by pathologic examination of the excised tissue, it seems likely that many of these resulted from pre-existing mediastinal infection of a non-specific character. Tuberculosis and syphilis are the most frequent specific infections which lead to fistula formation. The pathogenesis of tuberculous fistula is best explained on the basis of either erosion of caseating or calcified lymph nodes or rupture of cold abscess into the tracheobronchial tree and esophagus. In the absence of mediastinal tuberculous lymphadenitis, I have not observed ulcerative tracheobronchial tuberculosis to lead to fistulous communication with the esophagus. Tuberculosis of the esophagus is a rare disease. Syphilis may involve any part of the respiratory tract or esophagus and in its tertiary stage

TABLE II
ETIOLOGY OF THIRTY-ONE REPORTED CASES OF TRAUMATIC ESOPHAGORESPIRATORY FISTULA

Etiology	No. of Cases
Non-penetrating wound	9
Dilatation of stricture of esophagus	5
Foreign body	4
Penetrating wound	3
Operative	3
Chemical (lye)	3
Incomplete data	4

TABLE III
ETIOLOGY OF NINETY-THREE REPORTED CASES OF ESOPHAGOTRACHEOBRONCHIAL FISTULA

Etiology	No. of Cases
Traumatic	31
Undetermined	19
Tuberculosis	14
Diverticulum of esophagus	14
Syphilis	13
Empyema	1
Actinomycosis	1

may produce fistulas by necrosis and ulceration of gumma. Syphilitic aneurysms less frequently lead to fistula formation by pressure necrosis [5,8]. Two case reports of esophagorespiratory fistula caused by actinomycosis appear in the literature [10,12].

Traction diverticulum of the esophagus was responsible for fistula formation in fourteen cases. The development of a communication with the respiratory tree seems to be a relatively infrequent complication of diverticulum of the esophagus. Mellins [8] was unable to find any communication with the respiratory tract in twenty-three cases discovered during a study of 15,000 consecutive autopsies at Johns Hopkins Hospital. Although traction diverticula can be attributed usually to pre-existing periesophageal infection with subsequent contraction of scar tissue, it is not always possible to identify the initial infection. Whenever a specific infection can be identified, it is the result of tuberculous mediastinal lymphadenitis in the majority of instances. Traction diverticula may lead to fistulous communication with the respiratory tree by continued activity of the initial infection responsible for the diverticulum, by superimposed secondary infection with ulceration, or by erosion of calcified lymph nodes. The exact location of the opening of the fistula into the respiratory tree was recorded in twelve of the fourteen reported cases of this type. In eight of these, the esophagus communicated with a secondary bronchus which was usually in the lower lobe, and in four it communicated with the stem bronchus.

CLINICAL FEATURES AND DIAGNOSIS

Although the distressing symptoms associated with esophagorespiratory fistula are characteristic, patients may remain under observation for a period of a few weeks to twenty-five years without the benefit of a correct diagnosis and treatment. Patients with small fistulas may complain only of a chronic cough, but careful questioning frequently discloses that the cough is associated with ingestion of fluids. Such patients frequently have no difficulty ingesting solids. Small fistulas may be temporarily plugged by solid food which accounts for the periodic disappearance of symptoms. Occasionally febrile episodes secondary to inflammatory changes in the fistulous tract may lead to temporary alleviation of the symptom complex. The ingestion of fluids followed by strangling or severe paroxysms of coughing after a momentary asymptomatic pause is pathognomonic of esophagorespiratory fistula. If the fistula is large, solid foods produce the same symptoms and large particles of ingested food may be coughed up. Large fistulas may be associated with severe loss of weight, but some patients become accustomed to the inconvenience of the fistula and suffer little interference with nutrition. Many learn the advantage of swallowing in the dorsal recumbent position which tends to minimize the typical paroxysmal attacks of coughing and strangling (Ono's sign) [6]. Pulmonary hemorrhage is not an unusual symptom and it may terminate in death [9]. In some patients, repeated pulmonary hemorrhages have so overshadowed the symptoms of small fistulas that diagnosis has been delayed for as long as seven years [7]. Chronic cough, upper respiratory infections and repeated attacks of pneumonia are common occurrences. The fistula may lead to the development of lung abscess or bronchiectasis. A cough productive of a fetid or purulent sputum should suggest suppurative changes in the lung associated with the fistula.

Acute traumatic fistulas are notoriously difficult to recognize. Such an injury may be associated with either penetrating or non-penetrating trauma. There are frequently no immediate symptoms produced by a penetrating injury which would indicate disruption of the adjacent walls of the esophagus and trachea, but the path of the missile, abnormal dislocation of bone fragments and the presence of rapidly accumulating mediastinal emphysema or tension pneumothorax should arouse suspicion as to the possibility of such an injury.

Acute fistulas produced by non-penetrating injuries are even more prone to escape recognition. Adams and Mabrey [1] reported the only case that manifested immediate and unmistakable signs of fistulous communication between the trachea and esophagus. Usually two days to several weeks pass before symptoms of fistula are manifest, and this delay can be attributed to the temporary agglutination of the disrupted edges of the wounds of the trachea and esophagus. When the wounds re-open later as a result of infection or abnormal stress from coughing, symptoms of fistula develop. An acute fistula may be present but is easily overlooked in a patient with shock when ingestion of fluids is withheld and the

FIG. 3. Ingestion of lipiodol delineates the esophagotracheal fistula at the level of the superior thoracic aperture. (From: COLEMAN, F. P. and BUNCH, G. H., JR. Acquired non-malignant esophagotracheobronchial fistula. *J. Thoracic Surg.*, 19: 542, 1950.)

cough reflex mechanism is supressed. Of the nine reported cases of esophagorespiratory fistula caused by blunt force, an early pneumothorax developed in only one patient. Rapidly developing mediastinal emphysema, distortion and broadening of the mediastinal shadow, and inability to allay a rapidly accumulating pneumothorax by closed intercostal tube drainage of the pleural cavity may well indicate rupture of a large bronchus or an acute esophagorespiratory fistula.

The diagnosis of esophagorespiratory fistula is based on the history, physical examination, roentgenographic studies and endoscopic examinations. Trauma to the chest, ingestion of foreign bodies or caustics and instrumentation of the esophagus may be followed immediately or later by characteristic symptoms of fistula. Long-standing communications between the esophagus and respiratory tree are usually non-malignant. Fistulas of short duration in the cancer age group are suggestive of malignant lesions. If dysphagia precedes the development of symptoms, it is suggestive of cancer of the esophagus. A fistula of tuberculous origin may be preceded by a protracted and indefinite illness.

During the physical examination the symptom complex of esophagorespiratory fistula can usually be demonstrated easily by having the patient ingest water. If the lungs are examined following the paroxysm of coughing, it is possible to detect at times the appearance of rales in the lungs (Ono's sign) [*6*]. The examination may reveal evidence of traumatic deformity of the chest, tuberculosis, syphilis or mycotic disease. Clinical signs of respiratory tract infection may consist of the presence of moist rales over a lobe or signs of advanced suppurative disease of the lung. A fistula does not invariably lead to chronic suppurative disease of the lung, for experience with six cases disclosed no evidence of permanent changes in the tracheobronchial tree. A positive tuberculin skin test or even a positive sputum for acid-fast organisms is not necessarily indicative of a fistulous tract of tuberculous origin, but such disclosures must be reckoned with in the treatment of the patient. A positive serologic test for syphilis must also be therapeutically considered, but even closure of a fistulous tract under antisyphilitic therapy does not necessarily indicate that the fistula is of syphilitic origin. Cultures from indolent wounds of the cervical region, the chest wall or mediastinum in patients with esophagorespiratory fistula should be studied carefully for mycotic organisms.

Roentgenographic studies consist of a routine posteroanterior film of the chest, roentgenograms following the ingestion of contrast media and bronchograms. The latter study is performed in those patients with suspected irreparable damage to the tracheobronchial tree. Frequently barium sulfate is administered prior to the establishment of a diagnosis; however, ingestion of iodized oil is preferable because it is less irritating to the tracheobronchial tree. Suitable roentgenograms and fluoroscopy following ingestion of opaque oil will usually delineate the course of the fistulous tract. Ingested oil will occasionally spill over into the trachea from the pyriform sinuses, and to avoid confusion it is best to place a catheter in the upper cervical esophagus and administer the opaque oil by this method. (Fig. 3.) This procedure is of special value in the patient in

whom circumstances indicate the likely presence of an acute traumatic esophagorespiratory fistula. Bronchiectasis must be excluded by adequate bronchograms as its presence alters the surgical treatment of esophagorespiratory fistula.

Endoscopic examination of both the esophagus and tracheobronchial tree may be necessary for the establishment of the correct diagnosis when circumstances point to the likely presence of a recent traumatic communication between the esophagus and trachea. In the immediate post-traumatic period and even in the presence of shock, it is possible to identify traumatic disruption of the adjacent walls of the esophagus and trachea at a time when it is safe to proceed with direct surgical closure. Endoscopic examinations in the past have been reserved for those patients with long-standing fistulas. In such patients, esophagoscopy reveals the fistula to lie usually in the anterolateral wall of the esophagus. The opening may be easily overlooked due to its slit-like configuration. A bechic blast can be felt when the tip of the esophagoscope is proximal to the orifice of a large fistula. Biopsy is indicated in papillomatous and ulcerative lesions. Bronchoscopy is of value in confirming the communication of the fistulous tract with the respiratory tree. The opening of the fistula lies in the membranous wall of the trachea or bronchus. It may not be possible to visualize the orifice of the fistula in a secondary bronchus; however, it can be localized by injecting methylene blue solution into the esophagus during bronchoscopy. The colored solution may be observed to well up into the involved segmental bronchus.

TREATMENT

Careful analysis of ninety-three reported cases of fistula shows that approximately two-thirds of the patients received no specific treatment. In the group of fifty-eight patients untreated by surgery, the outcome was stated in twenty-seven: sixteen died while hospitalized, six showed no improvement and five were cured. (Table IV.) The results in thirty-five patients treated by surgery are extremely gratifying. Twenty-seven of the thirty-five patients were cured, four died and four showed no improvement. (Table V.) It is interesting to note that only six cases of fistulas subjected to direct surgical repair were reported from 1916 to 1949, while twelve additional cases have been reported since the latter date.

Few patients with acute traumatic esophagorespiratory fistula will be seen sufficiently early to warrant immediate operative closure of the fistula because of diagnostic difficulties and the relatively short period of wound contamination. Mediastinal infection or extensive mediastinal emphysema may develop in such fistulas before they become manifest and external surgical drainage of the mediastinum may be necessary. Distortion of the mediastinum, extensive mediastinal emphysema and a rapidly accumulating pneumothorax which cannot be adequately decompressed with closed intercostal tube drainage should suggest laceration of the

TABLE IV
NON-SURGICAL TREATMENT IN FIFTY-EIGHT REPORTED CASES OF ESOPHAGOTRACHEOBRONCHIAL FISTULA

	No. of Cases	Results			
		Not Stated	Cured	Un-changed	Died
No specific treatment	50	31	2	4	13
Antisyphilitic therapy	7	0	2	2	3
X-ray therapy	1	0	1	0	0
Totals	58	31	5	6	16

TABLE V
SURGICAL TREATMENT IN THIRTY-FIVE REPORTED CASES OF ESOPHAGOTRACHEOBRONCHIAL FISTULA

	No. of Cases	Results		
		Cured	Un-changed	Died
Cauterization (endoscopic):*				
Chemical	5	4	1	0
Electric	1	0	1	0
Direct surgical repair:				
With preliminary gastrostomy or jejunostomy	8	8	0	0
Without preliminary gastrostomy	10	8	0	2
Pulmonary resection and closure of esophageal defect	5	4	0	1
Palliative gastrostomy	3	0	2	1
Drainage of coexisting lung abscess	2	2	0	0
By-pass of fistula and stricture of esophagus (esophagojejunostomy)	1	1	0	0
Over-all Totals	35	27	4	4

* All small fistulas.

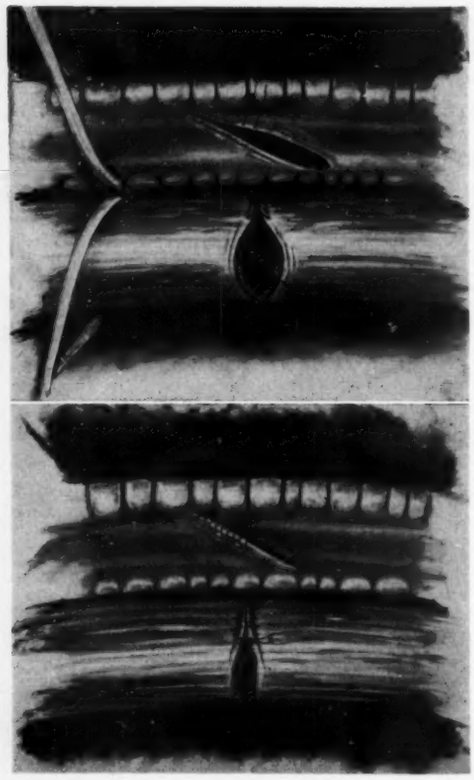

FIG. 4. Esophageal and tracheal defects following division of fistula. (*a*) First row of interrupted No. oooo silk sutures. (*b*) Second row of interrupted No. oooo silk sutures. (From: COLEMAN, F. P. and BUNCH, G. H., Jr. Acquired non-malignant esophagotracheobronchial fistula. *J. Thoracic Surg.*, 19: 542, 1950.)

trachea or major bronchus and possibly an acute esophagorespiratory fistula. More critical appraisal of this type of patient, supplemented if needed by bronchoscopy and the ingestion of lipiodol, will lead to possible recognition and early operative closure of acute traumatic esophagorespiratory fistulas.

Adequate evaluation of the patient for systemic disease and a careful study of the physical state of the fistulous communication is mandatory prior to consideration of operative closure. Fistulas associated with gummatous ulceration of the esophagus or tracheobronchial tree should receive vigorous antisyphilitic therapy. If active pulmonary tuberculosis is present or presumptive evidence of tuberculosis exists in a granulating fistulous tract, a preliminary course of antimicrobial therapy should precede direct surgical closure. Clerf [3] has shown that it is possible to close small fistulas (3 mm. or less in diameter) by endoscopic cauterization with silver nitrate or sodium hydroxide. Small fistulas and fistulas associated with exuberant granulation tissue or acute inflammatory reaction should receive local cauterization and antibiotics in expectation of cure or alleviation of local infection preparatory to later surgical closure. This can be determined by careful endoscopic study of the fistulous tract. Smooth epithelized communications are suitable for immediate and direct surgical closure and seldom respond to local cauterization.

Preoperative preparation of the patient consists of correcting protein depletion, vitamin deficiencies and secondary anemia, and restoring the contracted blood volume. Serious states of nutritional deficiency may exist and it may be necessary to perform preliminary jejunostomy or gastrostomy. Jejunostomy seems preferable in that it prevents regurgitation spill-over into the respiratory tree. In my experience with six patients gastrostomy or jejunostomy did not have to be performed, and the nutritional needs of the patients were met by means of gastric gavage through an indwelling Levin tube. Large fistulas developing a few days to several weeks after trauma may be handled best by jejunostomy and tracheostomy. A survey is made of the cardiac and renal function. Preoperatively, intramuscular and aerosol penicillin is administered for a period of three or more days.

Caldwell [2] has directed attention to the problem of anesthesia in acquired communications of the esophagus with the respiratory tree. Large fistulas communicating with the trachea are of little concern to the anesthetist, providing there is sufficient space for the insertion of an inflatable balloon between the carina below and the fistulous orifice above. If the fistula in the terminal trachea does not permit the interposition of a balloon between it and the carina, a snugly fitting intratracheal tube without a balloon will likely prove satisfactory [2]. No difficulty with routine intratracheal intubation anesthesia has arisen when the fistula communicated with a stem or secondary bronchus.

Direct surgical repair of an acquired esophagorespiratory fistula may require a cervical, a cervical combined with anterior mediastinal, or a transpleural approach. Fistulas located in the region of the superior thoracic aperture require a cervical approach combined with either a median sternotomy or resection of the inner third of the clavicle and the anterior portion of

the upper two ribs. Fistulas complicated by bronchiectasis or lung abscess may require pulmonary resection regardless of the site of the communication of the fistula with the tracheobronchial tree. It is always necessary to close the defect in the esophagus, whereas the site of the communication of the fistula with the respiratory tree may be removed with the resected lobe or lung. Fistulas located in the trachea or proximal stem bronchi are best approached by thoracotomy on the right. The azygos vein is divided and turned downward, and an excellent exposure of the proximal stem bronchi and terminal trachea can be obtained. The esophagus is mobilized above and below the fistulous communication with the respiratory tree. If the fistulous tract is of sufficient length after its division, a portion may be excised for pathologic study. The defects in the esophagus and trachea or bronchus are closed by one of the conventional methods of suture. Preferably, the trachea or bronchus is closed with two layers of interrupted No. 0000 silk sutures, a proximal longitudinal mattress suture and a distal simple on-end suture. The esophageal defect is closed in a transverse plane using two rows of interrupted No. 0000 silk sutures. Simple through and through sutures approximate the walls of the defect and the knots are tied within the lumen of the esophagus. The second layer consists of mattress sutures which approximate the muscular wall over the first layer of sutures. (Fig. 4.) The repaired defects are protected by the interposition of adjacent tissue. Closed intercostal tube drainage is established and connected to an underwater seal.

The postoperative care consists of detailed attention to evacuation of tracheobronchial secretions, removal of intercostal drainage tube in forty-eight hours (transpleural operation), parenteral penicillin and streptomycin, general supportative measures and fluids by mouth on the fifth postoperative day.

SUMMARY

This report is confined to a study of ninety-three reported cases of acquired non-malignant esophagorespiratory fistula.

The fistulas are classified on the basis of etiology. The known causes of acquired benign esophagorespiratory fistula are trauma, infection and diverticula of the esophagus. Trauma is the most frequent cause of this type of fistula, and there is a correlation between the type of trauma and location of the fistulous orifice in the respiratory tree. The most common specific infections leading to fistulous communications are tuberculosis and syphilis. Traction diverticula of the esophagus show a high incidence of communication with the secondary bronchi.

Development of a communication between the respiratory system and esophagus invariably leads to characteristic symptoms. Ingestion of fluids is followed by strangling or severe paroxysms of coughing after a momentary asymptomatic pause. Diagnosis is based upon history, physical examination, roentgenographic studies and endoscopic examinations.

Patients who have fistulas associated with active tuberculosis or syphilis should receive antibiotic therapy. Patients who have small fistulas and fistulas associated with exuberant granulation tissue should receive local cauterization and antibiotics in expectation of cure or alleviation of the local infection preparatory to later surgical treatment. Patients who have fistulas complicated by bronchiectasis or lung abscess frequently require pulmonary resection in addition to closure of the esophageal defect. Smooth epithelized communications unattended by suppurative disease of the lung are suitable for immediate and direct surgical closure.

The results of surgical treatment either by direct surgical repair or by closure of the esophageal defect with pulmonary resection are extremely gratifying. Only six patients treated by these methods were reported in the literature over a thirty-two-year period, 1916 to 1949, while nineteen additional cases have been reported in the English literature since the latter date.

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Intubation of the Esophagus

ITS ROLE IN PREVENTING ASPIRATION PNEUMONIA AND ASPHYXIAL DEATH

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DESPITE the fact that statistics prove aspiration is a leading cause of operating room deaths (Table I), sufficient attention has not been devoted by surgeons, obstetricians and anesthesiologists to the development of easily applied methods for the prevention of this catastrophe.

It is the purpose of this paper to focus attention on the conditions which predispose to aspiration of food or fluid and to point out that aspiration occurs much more frequently than is generally realized. (Tables II and III.) Emphasis will be placed on the technic of esophageal intubation developed by the authors for the prevention of pneumonia and asphyxia due to aspiration. Three representative cases in which this technic was used will be presented.

TABLE I
THE FREQUENCY OF ASPIRATION AS A CAUSE OF DEATH

Cole and Corday [1]	Reported 132 cases of cardiac arrest occurring in the operating room from all causes; in 21 cases death was due to an unclearable airway; of these, 11 were due to aspiration of vomitus (52.4%); all cases of cardiac arrest due to aspiration of vomitus (11) were irreversible
Hingson, Holden and Barnes [2]	Reported that aspiration of vomitus was responsible for 12 of 41 anesthetic deaths
Hingson and Hellman [3]	Found aspiration of vomitus responsible for 161 deaths of 331 maternal mortalities resulting from anesthesia

Basic Considerations. The larynx by its sphincteric action protects the lower respiratory tract from contamination by foreign substances. General anesthesia, coma or debility, by obtunding laryngeal reflexes, exposes the

TABLE II
BRONCHIAL ASPIRATION OF VOMITUS: DISTRIBUTION AMONG DISEASES ESSENTIALLY SURGICAL, 1929–1938*

Stomach and duodenum (perforation, gastric resection, infarction)	22
Biliary tract and liver (cholecystectomy, cholecystogastrostomy, carcinoma, cirrhosis)	18
Hemorrhagic infarctions of the small or large bowel (embolism, thrombosis)	11
Obstruction of bowel (adhesions, volvulus, incarceration)	10
Prostate gland (enucleation and resection)	10
Female genitalia (carcinoma, fibromyoma, pregnancy)	9
Cerebral neoplasm (glioma and carcinoma metastasis)	9
Bacteremia (lacerations of skin, phlegmons, lymphangitis)	9
Colon and rectum (resection of carcinoma)	9
Pancreas (necrosis, carcinoma)	6
Urinary bladder (carcinoma)	5
Fracture of cranial bones	3
Total	121
Total No. of necropsies 1,074 Instances of aspiration 169	Includes surgical and non-surgical patients

*Reproduced from IRONS, E. E. and APFELBACH, C. W. Aspiration bronchopneumonia. *J. A. M. A.*, 115: 584–586, 1940.

patient to the danger of aspiration and its complications, namely, asphyxial death, pneumonia, pulmonary edema or atelectasis. The latter may lead to bronchiectasis or lung abscess. Aspiration will occur even in the presence of a normal laryngeal mechanism

TABLE III
BRONCHIAL ASPIRATION OF VOMITUS: DISTRIBUTION AMONG DISEASES ESSENTIALLY NON-SURGICAL*

Apoplexy	8
Uremia	7
Meningitis	6
Dysentery	6
Lobar pneumonia	4
Diabetic coma	2
Total	33

Total No. of necropsies.. 1,074
Instances of aspiration... 169
} Includes surgical and non-surgical patients

* Reproduced from: IRONS, E. E. and APFELBACH, C. W. Aspiration bronchopneumonia. *J. A. M. A.*, 115: 584–586, 1940.

when the patient's demand for oxygen is so great that the glottis opens despite the presence of regurgitated material in the pharynx. This is most apt to occur when large volumes have been regurgitated. In general, whether or not aspiration occurs depends on the following factors: (1) the volume of material regurgitated, (2) the effectiveness of the vomiting mechanism, (3) the activity of the sphincteric action of the larynx, (4) the patient's demand for oxygen and (5) the effectiveness of the cough reflex in clearing the respiratory tract of foreign material.

Conditions Which Predispose to Aspiration:

I. During anesthesia:
   - A. "Full stomach" which may be due to recent ingestion of food or fluid, gastric atony, pyloric stenosis, intestinal obstruction, hemorrhage in the upper alimentary tract or post-tonsillectomy hemorrhage.
   - B. Esophageal diseases associated with stagnation of food or secretions, i.e., carcinoma, achalasia, diverticulum.
   - C. Lesions or diseases involving the upper respiratory tract, i.e., carcinoma of the tongue or pharynx, myasthenia gravis, poliomyelitis, paralysis of the vocal cords.
   - D. Immobilization of the mandible, i.e., temporomandibular fusion, intermaxillary fixation.

II. Comatose states—drug poisoning, injury to the brain, diabetic coma.

III. Debilitating conditions associated with gastric atony or dilatation.

IV. Tracheoesophageal fistula.

METHODS GENERALLY USED TO PREVENT ASPIRATION DURING ANESTHESIA, WITH THEIR DISADVANTAGES

Some physicians rely on normal gastric emptying times to decide whether or not it is safe to anesthetize the patient who has recently ingested food or fluid. This is dangerous because such conditions as trauma, shock, labor and use of narcotics impair gastric motility. Many physicians employ nasogastric suction to empty the stomach but in many instances this proves unsuccessful.

Anesthesiologists attempt to prevent aspiration by employing one of the following technics: (1) conduction anesthesia, which includes local, regional or spinal block, (2) endotracheal intubation of the conscious patient or (3) "crash induction" (extremely rapid induction into general anesthesia) with a barbiturate-relaxant mixture followed with quick endotracheal intubation.

Although a large number of anesthetists believe that patients under conduction anesthesia cannot aspirate because the technic does not depress pharyngolaryngeal reflexes, many patients have aspirated during its use. This has occurred most often in patients who have regurgitated large volumes of food or fluid and in debilitated or toxic patients with weakened pharyngolaryngeal reflexes. High spinal anesthesia, when used, predisposes to aspiration by reducing the patient's ability to cough and to project his vomitus. When, as often happens, patients under conduction anesthesia require supplementary general anesthesia, the safeguard of this technic (active pharyngolaryngeal reflexes) vanishes.

Endotracheal intubation of the conscious patient may be difficult even for the expert and does not protect against aspiration unless the cuff or tube fits snugly at all times against the wall of the trachea. It must be realized that a large number of patients are anesthetized by persons who are not trained endoscopists and

that therefore these patients will not receive the benefit of this technic.

"Crash induction" often produces profound circulatory depression which may prove fatal to the poor-risk patient; and if immediate intubation is not accomplished, the need for positive pressure may arise while the patient is regurgitating.

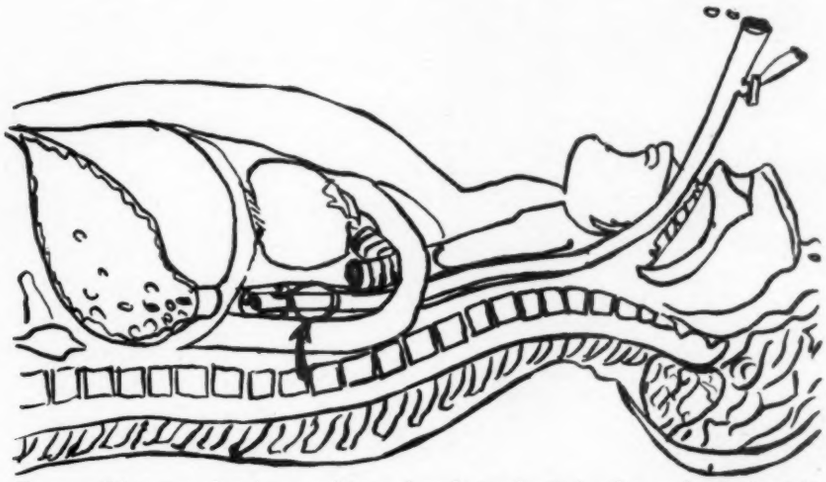

FIG. 1. Diagram showing position of endoesophageal tube and relationship of inflated balloon to adjacent structures. Inflated balloon (*arrow*) assures that all gastric contents pass to the outside via the tube. Since the pharynx is kept free of gastric contents, aspiration cannot take place.

ESOPHAGEAL INTUBATION

The authors [4] have developed esophageal intubation as a method of preventing aspiration. This technic is easily applied and establishes an effective barrier between the alimentary and respiratory tracts. (Fig. 1.) The technic is primarily indicated in the anesthetic management of the patient who is prone to aspirate and in the emergency treatment of the comatose patient. When used in the comatose patient, in addition to preventing aspiration of gastric contents it eliminates the hazard of flooding the patient's lungs during lavage and gavage. It can also be used to empty the stomach and the esophagus when other measures have failed.

Advantages. The insertion of an esophageal tube requires no greater skill than the passing of a Levin tube. Esophageal intubation, by removing the restrictions which the threat of aspiration imposes upon the choice of anesthetic agent and technic, allows the anesthesiologist a wider range of agents and technic from which to choose. Esophageal intubation usually empties the dilated stomach and so eliminates it as a source of pernicious cardiovascular reflexes, permits better pulmonary excursions and, in the surgical patient, also helps to insure good exposure.

The conscious patient will tolerate an esophageal tube for a much longer period of time than he will an endotracheal tube. The endoesophageal tubes are not expelled by the patient because the force of vomiting is rapidly dissipated through their large lumens.

An important factor recommending the use of this technic is the absence of deleterious effects. Electrocardiographic studies have revealed only mild transient tachycardia which lasted from one to three minutes. Radiographic and bronchoscopic examinations made while the esophageal tube was *in situ* did not show tracheal compression, and esophagoscopic examinations performed following esophageal intubation did not demonstrate any mucosal damage.

Technic. With the possible exception of children and uncooperative adults,* all patients should be intubated before general anesthesia is begun.

Topical anesthesia is applied to the oropharynx if the gag reflex is exaggerated. The selected tube is introduced into the pharynx and advanced gently into the esophagus for

* These patients usually require light general anesthesia prior to esophageal intubation.

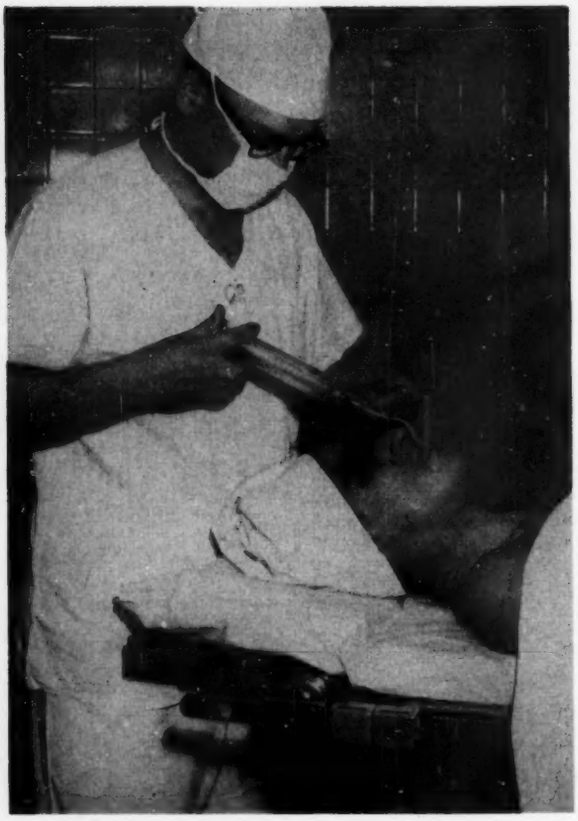

FIG. 2. Photograph of conscious patient with endoesophageal tube *in situ*. Balloon of tube is being inflated with 15 cc. of air. The proximal end of the tube protrudes from the oral cavity. Passage of the endoesophageal tube causes no greater discomfort to the patient than does the insertion of a Levin tube.

the recommended distance, and the cuff is then inflated. (Fig. 2 and Table IV.)

When the oral route cannot be used because of anatomic (e.g., bilateral temporomandibular fusion) or other reasons, esophageal intubation can be accomplished through the nose.

Once the tube is in place, the patient may be anesthetized with whatever agent and technic are indicated. Esophageal extubation should not be done unless it is believed that the danger of aspiration is no longer present.

The equipment necessary for esophageal intubation is shown in Figure 3.

CASE REPORTS

CASE I. A fifty-three year old woman was admitted to the emergency room of the Columbus Hospital in respiratory arrest after having ingested a large number of capsules containing barbiturate and tranquilizer drugs. Physical examination revealed a markedly cyanotic woman in shock and in complete areflexia. Following the insertion of an endotracheal tube and the institution of positive pressure respirations, a No. 32 French endoesophageal tube was passed. After large amounts of gas, fluid and partially digested food were recovered, the

TABLE IV
GUIDE TO SELECTION OF ESOPHAGEAL TUBE

Age	Caliber (French)	Distance Inserted from Incisor Teeth (cm.)*	Cuff (cc.)
Neonatal...	16	15 (from gum margin)	0
Infant–			
1 yr.....	16–18	18	0
2–4 yr.....	18–24	22	0–5
5–11 yr....	24–30	26	5–10
12–14 yr...	30–32	28	10–15
15 yr...	30–32	32	10–15
16 yr.–adult	30–32	35	15–20

* Based on studies of the esophagus by William Lerche, M.D.

stomach was lavaged by means of a gastric tube introduced through the endoesophageal tube. Tracheostomy was performed because the patient was expected to remain in coma for a long time. Her respirations were initiated and sustained by means of a Reiter electrostimulator. She regained consciousness in forty-eight hours and made an uneventful recovery. Repeated chest x-rays taken during the first forty-eight hours showed normal lung fields.

Comment: This case illustrates the way in which esophageal intubation used in the emergency treatment of the comatose patient eliminates the threat of massive contamination of the respiratory tract by gastric contents or by lavaging fluid.

CASE II. A seventy-six year old obese woman was admitted to Columbus Hospital on April 3, 1956, because of acute heart block with Adams-Stokes syncopal attacks. Her blood pressure was 80/40, pulse 30 and respirations 40. She was cyanotic and dyspneic, vomited repeatedly and had marked abdominal distention. An electrocardiogram showed an auricular rate of 100, a ventricular rate of 30 and auricular-ventricular block. X-ray revealed enormous gastric dilatation. (Fig. 4.) The next day she was semi-stuporous and in critical condition. Inasmuch as gastrointestinal intubation with

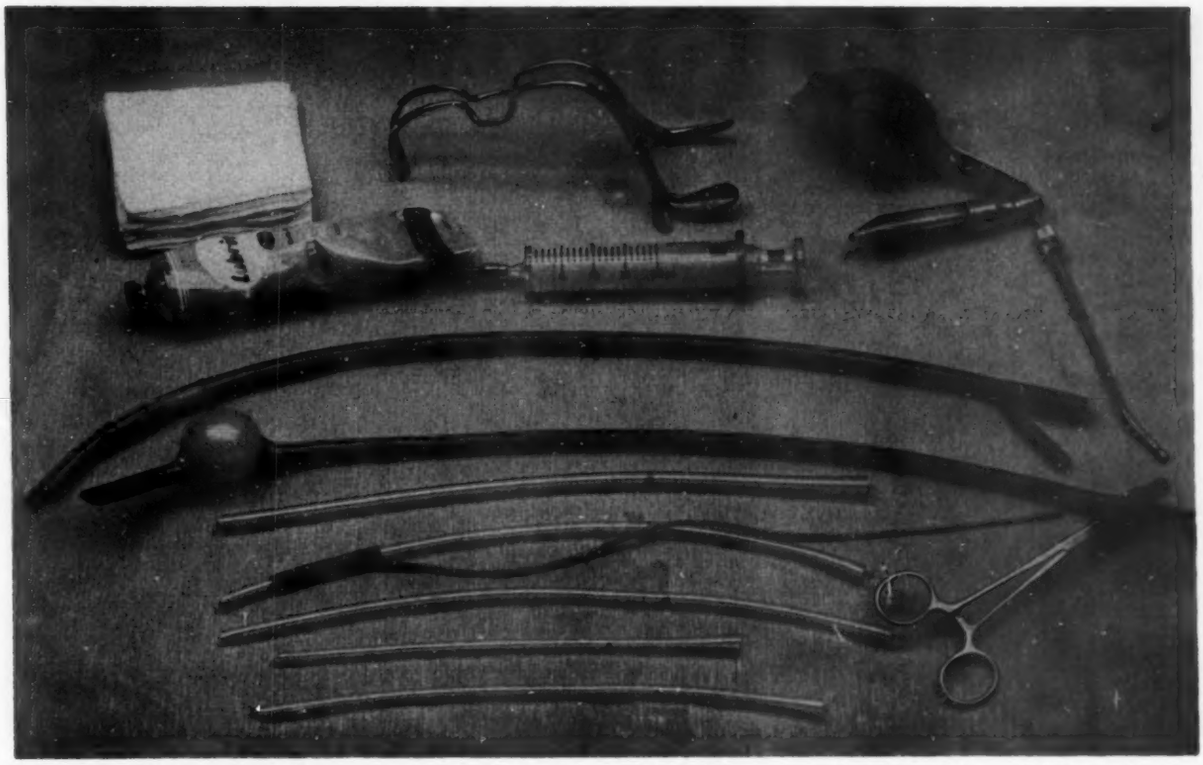

FIG. 3. Equipment for esophageal intubation. Note that each tube* bears a mark. When this mark is opposite the incisor teeth, it signifies that the tube has been inserted into the esophagus for the recommended distance. The mouth gag is used to keep the mouth open during induction in patients who require general anesthesia and to prevent biting of the tube.

Levin and Miller-Abbott tubes had failed to relieve the distention, we were called upon to insert an endoesophageal tube. Large amounts of gas and fluid escaped through the tube with explosive force and the patient improved dramatically. She became alert, was able to breathe with ease and her pulse rate rose from 30 to 90. An electrocardiogram taken shortly after the procedure revealed an auricular rate of 100, a ventricular rate of 100 and sinus tachycardia. X-rays showed absence of gastric dilatation. (Fig. 5.)

Comment: This case points out that esophageal intubation was successful in relieving gastric dilatation after the commonly used Levin and Miller-Abbott tubes had failed. The electrocardiographic record showed that the auricular-ventricular block, which was believed to be due in part to vagal reflexes emanating from the distended stomach, was immediately corrected when the stomach was deflated.

CASE III. An intoxicated young adult man was brought to the operating room for emergency reduction of bilateral compound fractures of the mandible and intermaxillary wiring. After a No. 28 French nasoesophageal tube was passed, light general anesthesia was administered employing the nasotracheal technic. During emergence from anesthesia the patient vomited through the nasoesophageal tube. Half an hour after the completion of surgery it was thought safe to remove the nasotracheal tube because the patient was awake. The tube was replaced with a nasopharyngeal airway. The esophageal tube was removed four hours later because the danger of aspiration had seemingly passed, i.e., the patient was fully conscious and had not vomited, retched or felt nauseated during this period of time. Recovery was uneventful.

Comment: It is generally recognized that the danger of asphyxia due to aspiration of vomitus is great during emergence from anesthesia when the patient's mouth cannot be opened because of either surgery or disease. We have shown in this case how nasoesophageal intubation was used to avoid this danger.

* Manufactured by the Davol Rubber Co., Providence, R. I.

FIG. 4. Case II. X-ray of abdomen showing tremendous dilatation of the stomach.

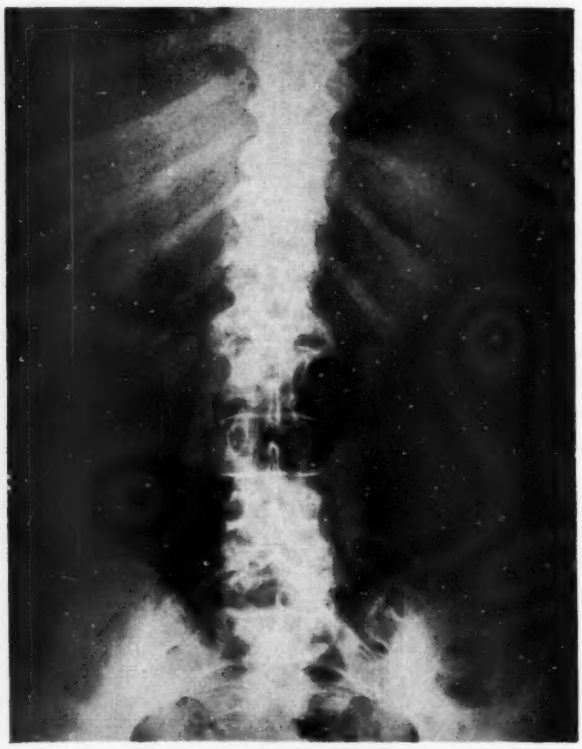

FIG. 5. Case II. X-ray of abdomen taken after esophageal intubation, showing absence of gastric dilatation.

SUMMARY

Attention has been called to the frequency with which aspiration occurs in the operating room, to the conditions which predispose to aspiration, and to the deficiencies of the methods generally used to prevent aspiration.

The authors have discussed their technic of esophageal intubation for the prevention of aspiration, with its advantages.

Three cases have been reported to demonstrate the use of esophageal intubation (1) during the emergency treatment of the comatose patient, (2) as a means of emptying the stomach when other measures have failed, and (3) during anesthesia.

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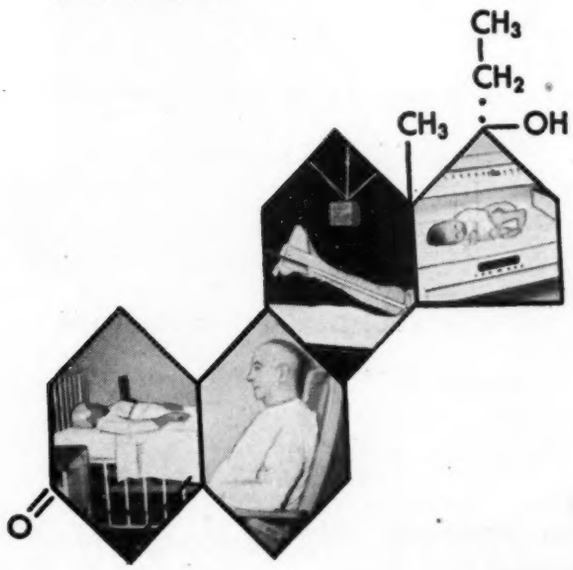

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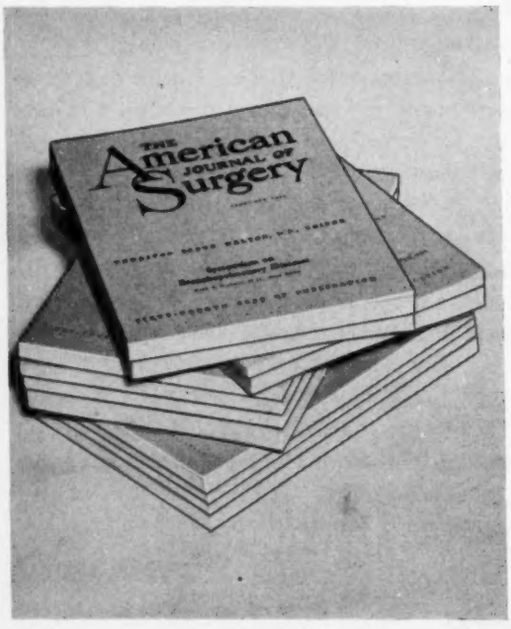

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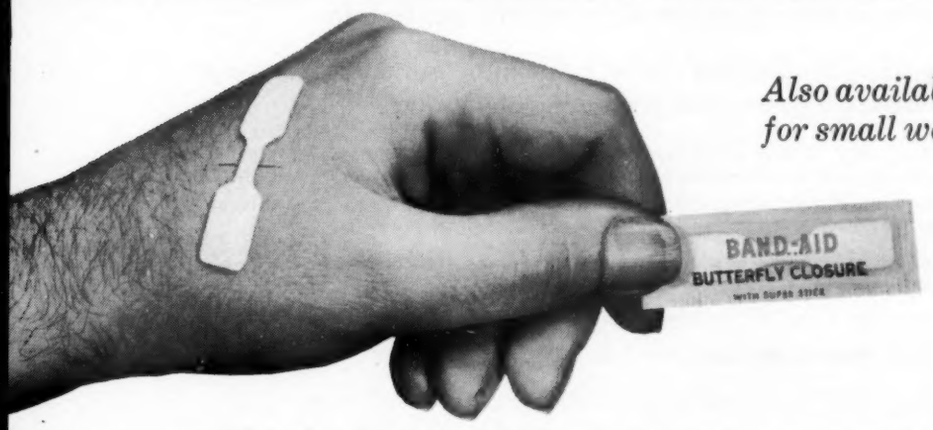

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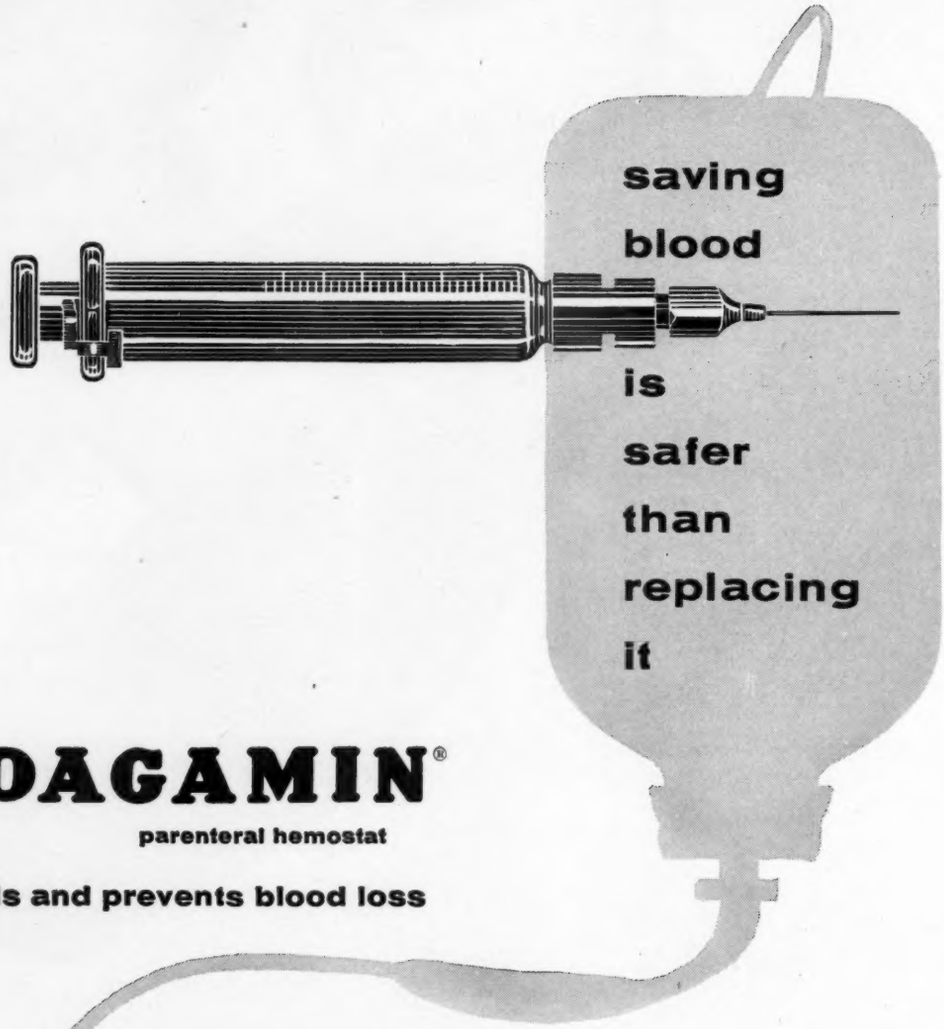

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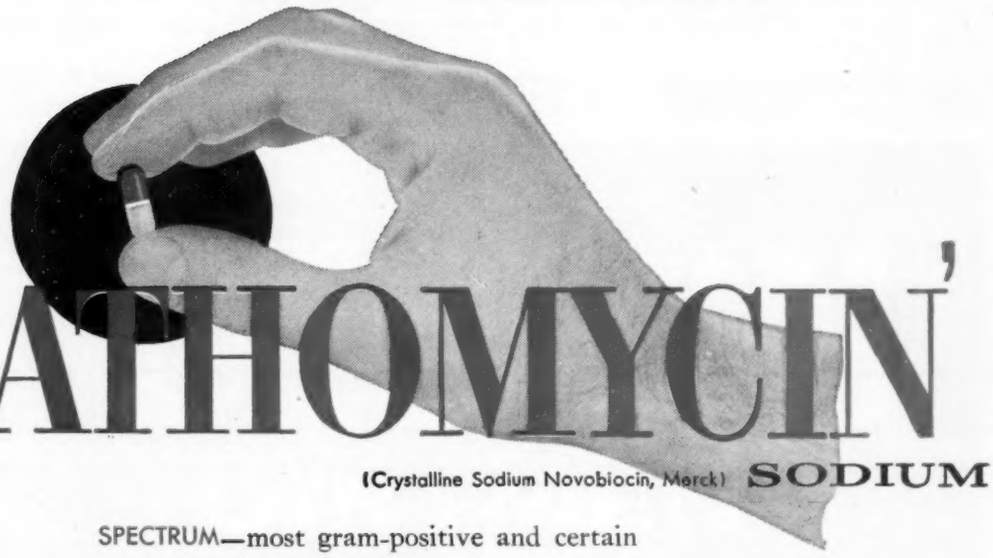

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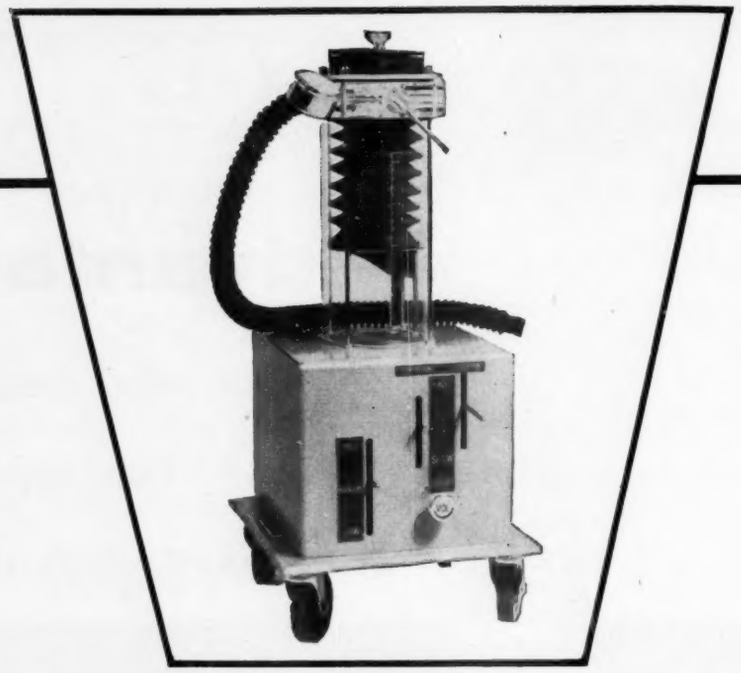

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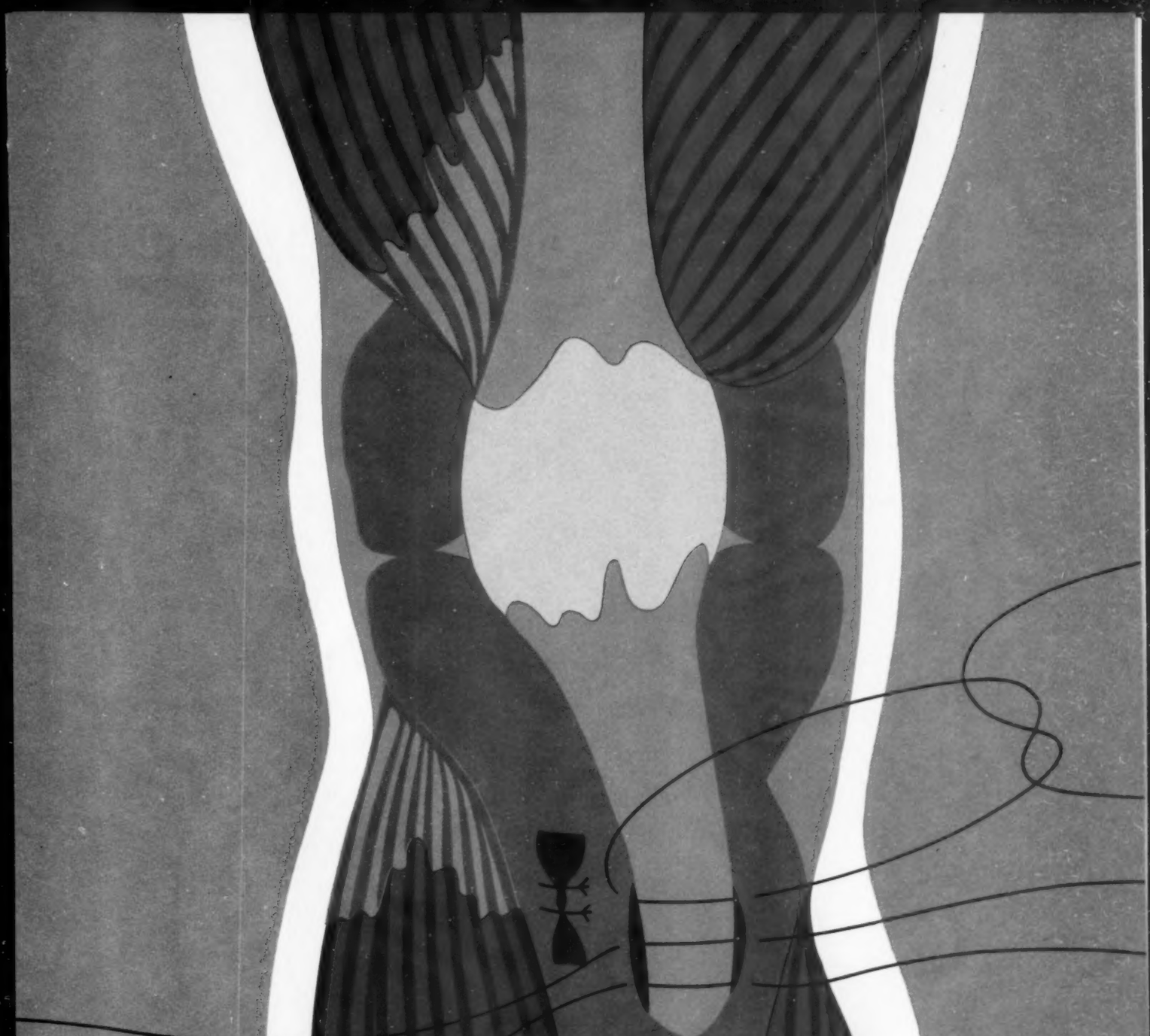

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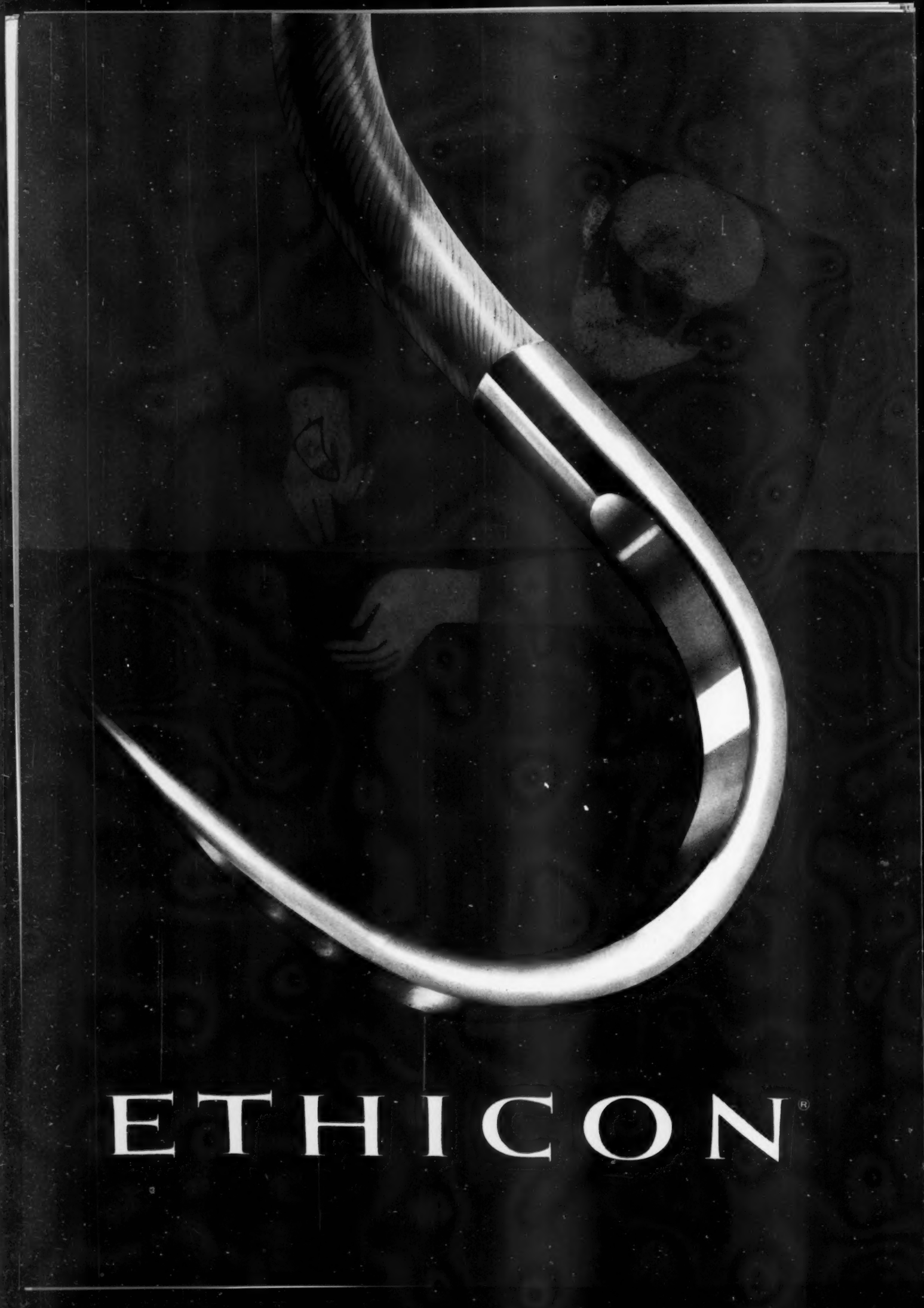
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to improve protein nutrition before and after surgery

Cerofort®

tablets

(Critically essential L-lysine with therapeutic amounts of all essential vitamins)

Major surgery inevitably leads to an accelerated breakdown and loss of body protein. In severe cases this may amount to between two and three pounds a day.[1]

Unless these losses are balanced by an adequate supply of high-quality proteins before and after surgery, convalescence is prolonged and wound healing is delayed.[2]

All too often, however, pre- and postoperative patients are unwilling or unable to eat enough high-quality protein to satisfy their need for increased amounts of essential amino acids in the right proportions. Instead, the "soft" diet they select is largely made up of vitamin-poor foods and cereal proteins, relatively deficient in the essential amino acid, L-lysine.

When you prescribe Cerofort Tablets for your surgical patients, you supply them with physiological amounts of L-lysine and therapeutic amounts of all essential vitamins. L-lysine raises the biological value of cereal proteins to that of high-quality animal proteins; the vitamins play vital accessory roles in protein synthesis and wound healing.

Also available:

Cerofort® elixir

(L-lysine with essential B vitamins)

TO POTENTIATE PROTEIN SYNTHESIS

- *in the elderly*
- *the convalescent*
- *the adolescent*
- *the growing child*

References: 1. Elman, R., and Cannon, P. R.: in Clinical Nutrition, Jolliffe, N., et al., eds., New York, Paul B. Hoeber, Inc., 1950, p. 183. 2. Pollack, H., and Halpern, S. L.: Therapeutic Nutrition, Washington, D. C., National Academy of Sciences, National Research Council, Publication 234, 1952.

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The normal skin has an acid pH between 4 and 6. This acid mantle acts as a protective barrier.

When the skin is washed with soap or detergents, or is exposed to chemicals, solvents, et cetera, the protective acid mantle is removed.

This exposes the unprotected skin to contact irritants and pathogenic organisms. It results in a rise in the skin pH above 7, provides a fertile field for development of harmful bacteria and fungi, and may result in various types of dermatitis.

Dome Acid Mantle returns the skin to its normal acid pH in a matter of seconds and holds it for hours. Both the creme and lotion are greaseless, stainless.

AVAILABLE—Acid Mantle Creme pH4.2 in 1 oz. tubes, 4 oz. and 16 oz. jars. Acid Mantle Lotion pH4.5 in 4 oz. squeeze bottles and 16 oz. bottles.

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CREME or LOTION-DOME pH4.2

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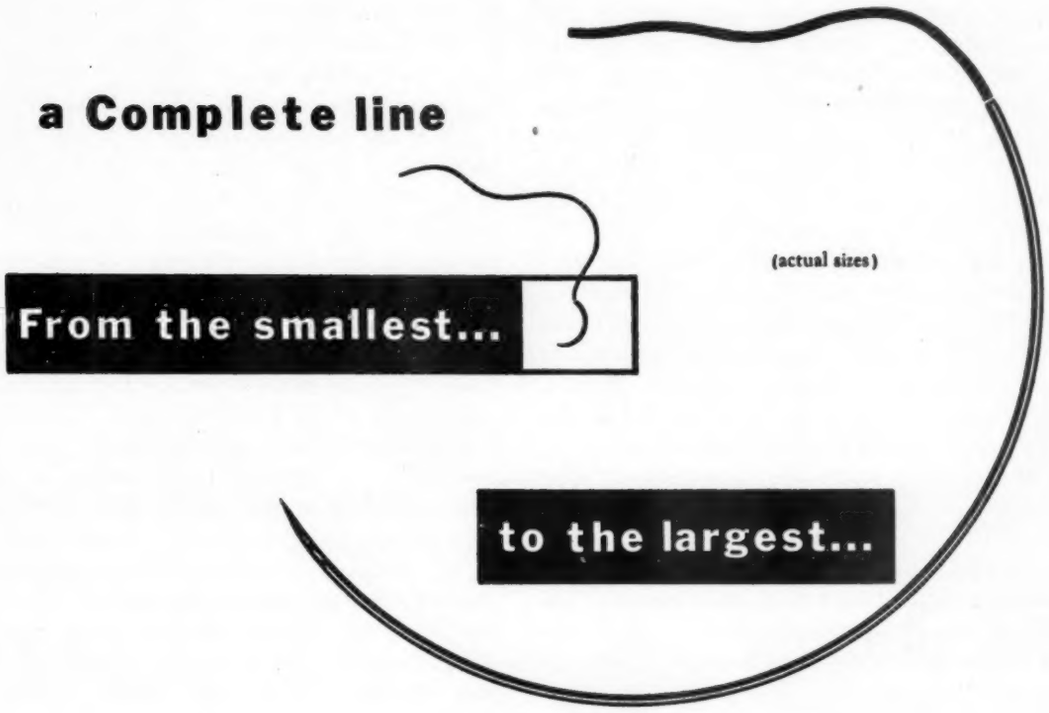

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NOTES AND QUOTES

As of January 1, 1956, operation of a cancer registry became a minimum requirement—in addition to (1) a Committee on Cancer and (2) a Cancer Consultation Service or a Cancer Consultation and Treatment Service—for survey, by the American College of Surgeons, of cancer programs in general and special hospitals conducting cancer clinics and cancer diagnostic clinics. Details of organization and operational procedures for a satisfactory cancer registry have been worked out by the Committee on Cancer of the American College of Surgeons and are published as a Manual for Registries and Cancer Clinical Activities by the Department of Professional Services and Accreditation, American College of Surgeons, 40 East Erie St., Chicago 11, Illinois, at 50 cents per copy. This twenty-page brochure contains pattern blanks for recording pertinent data, a Cancer Registry Abstract, a Year Book (Accession Register), and a Patient Index Card.

A cancer registry, to be accredited by the Committee on Cancer, American College of Surgeons, must contain the record of every cancer patient, private and public, inpatient and outpatient, admitted to the hospital. It includes a standardized abstract of each patient's clinical record and annual follow-up notes.

Operation of cancer registries should be extended beyond the special cancer institutions to include all facilities concerned with diagnosis or treatment of cancer and with the statistical study of cancer, at county, state and national levels. When this has been accomplished, workers in this field will have reliable statistical information upon which to base further research and improvements in the diagnosis, treatment, prognosis and control of cancer.

An international team of doctors, back from a month-long visit to Africa where they conducted pioneer blood pressure studies on giraffes, has come up with data that may help solve the problem of blackouts among high-speed aircraft pilots.

Conducted under a contract between Duke University and the U. S. Office of Naval Research, the project is the first intensive physiologic study of giraffes ever made. The six-man research team was composed of physicians from the United States, South Africa and Germany.

Working on a game preserve in the Union of South Africa, the doctors sought to answer a question that has puzzled scientists for half a century: How does the giraffe manage to pump blood from its heart all the way up a neck that may be as long as 12 feet? The answer seems to be high blood pressure.

Dr. James V. Warren, Duke Medical School professor and a member of the research group, said recently in an interview that the blood pressure of the animals studied was high enough to force blood to the brain. The giraffe's blood pressure may be three or four times that of a normal human being, he explained. Previously it was suspected that the giraffe might be equipped with a siphon-like mechanism to boost the blood on its long, upward journey.

The research relationship between giraffes and jet pilots stems from the fact that blackouts are caused by the heart's inability to pump blood uphill to the brain against terrific gravitational pressures—a situation comparable to the one that would exist if a man's neck were extended to a length of 10 feet or so.

In addition to taking blood pressures, the researchers measured blood volume and studied the pumping action and electrical activity of the giraffe's heart. The group also conducted respiratory studies that included measurements of air intake and oxygen consumption.

Detailed reports of the doctor's findings will be published in medical journals in the near future.

A FIVE year old boy whose heart action was purposely stopped for fifteen minutes by Duke Hospital surgeons during a major operation is now well on the road to recovery, it was reported recently.

Using a new low temperature and artificial "heart-lung" technic developed at Duke, the surgeons opened the boy's heart to repair a hole between the two heart chambers. The operation marks the longest time that a patient's heart action has been halted at Duke Hospital through the use of the new procedure announced in mid-1956.

The young patient underwent surgery for correction of a congenital interventricular heart defect. Before operation, his temperature was lowered from the normal 98.6°F. to 89°F. by surrounding his body with ice packs.

CONTINUED ON PAGE 48

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EXTRACTION

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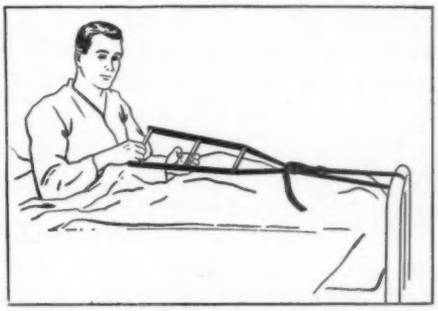

The New Posey "Patient Aid"

The new Posey "Patient Aid" is another rehabilitation product which encourages self-exercise and is a positive aid to the geriatric. The patient can raise himself without calling for the nurse.
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An extensive discussion giving details of the technique of regional analgesia may be found in our booklet "Sarapin, Injection Technique in Pain Control," and is available on request.

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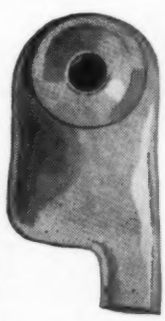

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NOTES AND QUOTES

Excellent health conditions prevailed among the American people in 1956, according to the statisticians of the Metropolitan Life Insurance Company. A continued favorable health record is expected in 1957.

For 1956, the death rate in the United States is indicated as 9.3 per 1,000 population, or on a par with that for 1955. The year of 1956 is the ninth in succession to show a national death rate below ten per 1,000 population.

A noteworthy feature of the mortality in 1956, the statisticians noted, is the exceptionally low death rate from tuberculosis—about nine per 100,000 population. This represents a decrease of approximately 5 per cent from the all-time low established the year before. As recently as a decade ago the tuberculosis death rate was four times its present level. A quarter century ago the rate was more than eight times what it is now, and it was more than twenty times as high at the beginning of the century.

THE AMERICAN MEDICAL ASSOCIATION at their Tenth Annual Clinical Meeting in Seattle, Washington, cited Ciba Pharmaceutical Products, Inc. for service to the medical profession through its presentation of the national television series, "Medical Horizons."

The President of the A. M. A., Dr. Dwight H. Murray, presented the citation before the association's House of Delegates assembled for its annual meeting. The citation, approved by the A. M. A. Board of Trustees, was accepted by T. F. Davies Haines, President of Ciba.

In accepting the A. M. A. plaque, Mr. Haines said, "The fact that you have awarded this citation to Ciba shows that we have in large measure succeeded in accomplishing what we set out to do when, two years ago, we began our series of television programs, which was in the words of your citation to 'give the American public a new insight into the work and achievement of the American doctor.'"

Poliomyelitis cases in the United States were little more than half the number recorded in 1955. By the end of 1956 about 15,500 cases of the disease were reported, compared with 29,000 the year before and with almost 58,000 in 1952. Some of the decrease in 1956 undoubtedly reflected the effectiveness of the Salk vaccine.

THE next scheduled examinations (Part II), oral and clinical, for all candidates for the American Board of Obstetrics and Gynecology will be conducted at the Edgewater Beach Hotel, Chicago, Illinois, by the entire Board from May 16 through 25, 1957. Formal notice of the exact time of each candidate's examination will be sent him in advance of the examination dates.

Candidates who participated in the Part I examinations will be notified of their eligibility for the Part II examinations as soon as possible.

The Zimmer Manufacturing Company, Warsaw, Indiana, announces the manufacture of a holder for two legs designed by Dr. Richard W. Zollinger of Columbus, Ohio. The leg holder has proved to be especially useful in the preparation of the lower extremities for venous strippings for various veins. It makes access to the short saphenous system easy, without repositioning the patient on the operating table; it is useful for other soft tissue surgery on the lower extremities, and it can be used for the holding of an extremity for approximation of a fractured patella.

THE Electrodyne Company of Norwood Massachusetts, announces a new electronic instrument, the Electrodyne E-11, the first instrument to provide detection and treatment of both cardiac arrest and ventricular fibrillation. This new product is a combination of two other Electrodyne instruments, the D-72 External Defibrillator and the Cardiac Pacemaker.

A report from the University of Wisconsin Tumor Clinic reveals that most cancer patients and their families do not fear the disease, want to know more about it, and think cancer education saves lives.

Drs. Robert Samp and Anthony Curreri surveyed 560 persons on their attitudes toward cancer education. The results of the survey indicate that persons most affected by cancer—the patients themselves and their families—believe cancer education is not being overstressed and is not resulting in "cancerphobia."

This is probably the first survey which does not rely on personal impressions to estimate the value of cancer education.

CONTINUED ON PAGE 50

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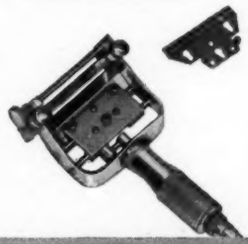

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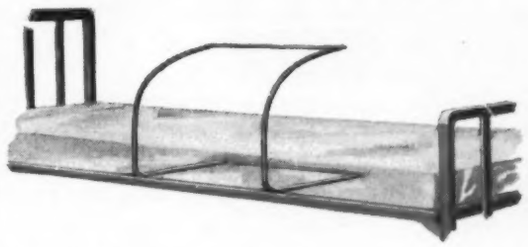

This Bed Cover Support can be placed at any part of the bed. It is secured between mattress and spring. It does not have to be moved in order to attend patient.

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Catalog will be sent upon request

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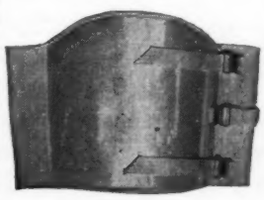
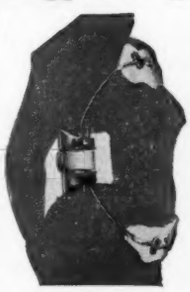


CAMP Spinal Hyper-extension Brace



A new washable and odorless Camp brace for positive hyper-extension in cases of compression fractures, osteoporosis with kyphosis, Marie-Struempell's disease and adolescent epiphysitis. Easily adjusted, rotating pads at sternal and pubic areas to eliminate pressure edges.

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NOTES AND QUOTES

"We realize that a study which includes only 560 persons is statistically inadequate for a conclusive report," Dr. Samp said. "Nevertheless, the striking unanimity of opinion on certain questions indicates definite trends of thought of those most affected by the disease."

JUNE, 1957, marks the tercentenary of the death of William Harvey, who discovered the circulation of the blood. This is of great historical importance to scientists not only in this country but also throughout the world. It is proposed to commemorate the occasion by holding an International Congress on Circulation from June 3 to 7, 1957, in the Royal College of Surgeons, London. The range of subjects covers every aspect of circulation and names of speakers who have already agreed to take part are appended in a preliminary program. Further information may be obtained at any time from the Secretariat at the aforementioned address, and a complete program, giving details of both the scientific and social activities, will be available in the near future.

The death toll from accidents in the United States was higher in 1956 than in the year before, a consequence of the rise in motor vehicle accident fatalities. Fatal injuries in the home and among workers have remained relatively unchanged in number. Homicides also have continued on the same level, while suicides have decreased somewhat.

"The excellent health record of the American people results largely from the rapid progress made in the various branches of the medical sciences and from the rise in the general standard of living," the statisticians concluded. "Further gains can be made by the fuller utilization of existing knowledge and by the development of new knowledge through research."

To provide better service and greater convenience to physicians and surgeons in New York City and the surrounding area, the George P. Pilling & Son Co. has established an office and display room at 4 West 56th Street. Mr. James P. Kelly is in charge of the office which maintains a complete display of instruments for bronchoesophagology, cardiac and thoracic surgery, eye, ear, nose and throat and general surgery.

Physicians, surgeons and hospital personnel are cordially invited to visit this show room to inspect the full line of Pilling instruments and equipment. The New York office is in direct telephone contact with the Philadelphia office and is therefore able to take care of special requirements.

In addition, the Pilling company has established resident representatives in the Los Angeles and Washington, D. C. areas for faster service to the profession in those areas.

Hopeful news in the war against cancer is reported by Metropolitan Life Insurance Company statisticians in the downward trend over the past ten years in the cancer death rate among women in middle life.

Among the insurance company's millions of industrial policyholders, mortality from all types of cancer was down by 13 per cent for women at ages forty-five to sixty-four over the ten-year period. One of the brightest features of the insurance experience is the decline by one-third in the death rate from uterine cancer at these ages.

This favorable trend is attributed to the prevention or correction of injuries sustained during childbirth, the intensified search for cancer in its early stages, and prompt treatment by surgery or radiation.

THE Eleventh Annual M. D. Anderson Hospital and Tumor Institute Symposium on Fundamental Cancer Research will be held on March 7, 8 and 9, 1957, at The University of Texas M. D. Anderson Hospital and Tumor Institute in the Texas Medical Center, Houston. The general topic for the Symposium is "Viruses and Tumor Growth." The first day of the Symposium will consist of open house to all symposium members, and presentation of annual progress reports on certain current research projects will be made by the principal investigators of M. D. Anderson Hospital.

Iowa and New Hampshire have the highest proportion of elderly people in their population. In both states 11 per cent of the total population—about one person in nine—is sixty-five years old or older. New Mexico and Arizona have the lowest proportion in the sixty-five year old or older bracket—New Mexico with 5.3 per cent and Arizona with 5.8 per cent.

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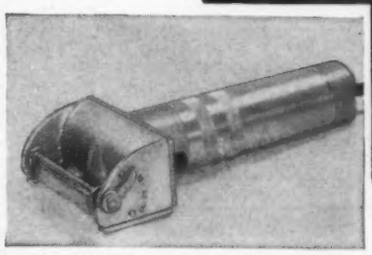

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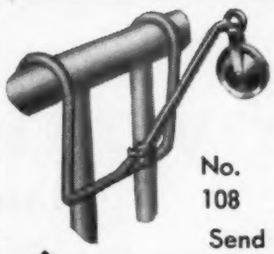

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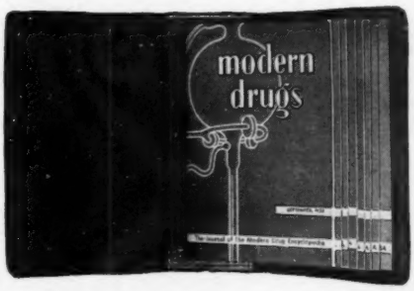

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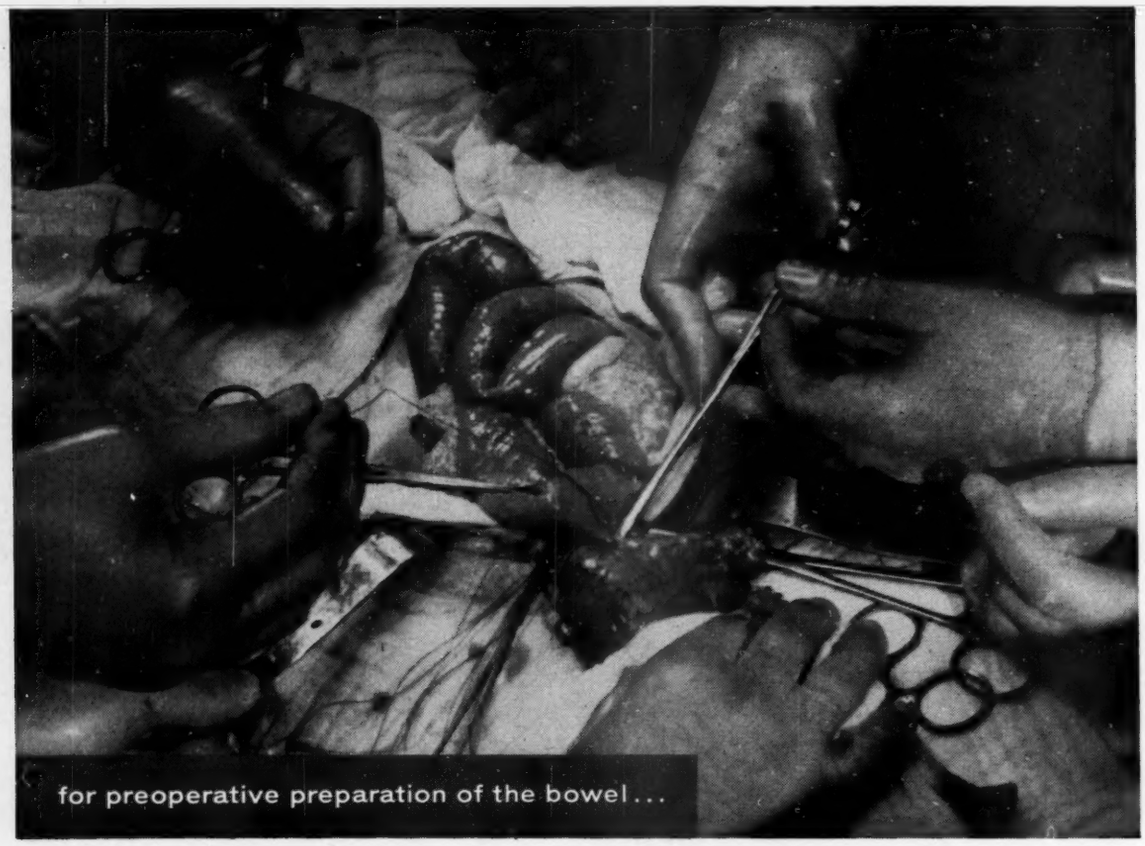

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FEBRUARY 1957

THE American JOURNAL OF Surgery

ADVERTISERS INDEX

Abbott Laboratories... *Insert Facing Page* 8
Aeroplast Corporation.....20–21
American Hospital Supply Corporation.. 4
American Meat Institute..... 36
American Sterilizer Company..... 11
Ames Company, Inc..... 1
Astra Pharmaceutical Products, Inc.....
Insert Facing Page 24
Bard-Parker Company, Inc..... 30
Baxter Laboratories, Inc..... 4
Cambridge Instrument Co., Inc..... 24
S. H. Camp and Company..... 49
Chatham Pharmaceuticals, Inc..... 38
Chesebrough-Pond's Inc..... 19
Cook County Grad. School of Medicine. 40
American Cyanamid Company..... 31
J. A. Deknatel & Son, Inc..... 45
De Puy Manufacturing Co., Inc..... 51
Desitin Chemical Company..... 41
Dome Chemicals, Inc..... 44
Duke Laboratories, Inc..... 44
Duxe Products..... 47
Eastman Kodak Company..... 15
Eaton Laboratories..... 6, 13
Ethicon Suture Laboratories, Inc.....
Insert Facing Page 42
Otis E. Glidden Co., Inc..... 51
High Chemical Company..... 47
Hoffmann-La Roche, Inc. *Insert Facing Page* 16
Kansas City Assemblage Co..... 51
Johnson & Johnson..... 37
Lederle Laboratories.....16, 34
Liebel-Flarshiem Co..... 26
Eli Lilly and Company..... 32
Little, Brown & Company..... 14
Mead Johnson & Company..... 10
Merck Sharp & Dohme..8, 25, 39, *Fourth Cover*
V. Mueller & Co..... 42
N. Y. Polyclinic Medical School & Hospital..... 40
Notes and Quotes.....46, 48, 50
Orthopedic Equipment Co..... 51
Murle Perry..... 49
Pfizer Laboratories, Division, Chas. Pfizer & Co., Inc..... 9, 23
J. T. Posey Company..... 47
The Radium Emanation Corp.....47, 49
Rajowalt Company..... 49
Royal Surgical Appliance Inc..... 51
H. W. Rutzen & Son..... 47
Rystan Company..... 18
Sanborn Company..... 35
Schering Corporation.....27–28–29
G. D. Searle & Co..... 33
Shampaine Company..... 17
J. Sklar Mfg. Co., Inc..... 22
E. R. Squibb & Son, Division of Matheson Chemical Corp..... 52
The Surgeon's Mart.....47, 49, 51
Edward Weck & Co., Inc..... 12
White Laboratories, Inc..... 43
Winthrop Laboratories..... 2
Wright Mfg. Co..... 49
Zimmer Manufacturing Co..... 49

A good start for safer bowel surgery

SULFATHALIDINE®

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The virtual "sterilization" of pathogenic bowel contents which follows preoperative administration of SULFATHALIDINE eliminates a major danger in intestinal surgery. Postoperatively, SULFATHALIDINE continues the suppression of dangerous pathogens, lessens the likelihood of peritonitis and abscess formation. Flatus is minimal; fungi and yeast do not overgrow their habitat; early healing is promoted. SULFATHALIDINE is an important adjunct in ulcerative colitis.

CREMOTHALIDINE® is a palatable suspension of SULFATHALIDINE. Each 30 cc. (1 fluidounce) contains 6.0 Gm. SULFATHALIDINE.

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